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INTELLIGENT PHYSIOLOGIC MODELING

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AN APPLICATION OF [redacted] TECHNOLOGY
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by

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ABSTRACT

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Table of Contents

List of Figures

List of Tables

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1. Introduction

This thesis describes a research project which applies knowledge based modeling techniques from artificial intelligence technology to certain aspects of medical education. In order to set the stage for details of the project, this chapter relates the recent history of medical education and the nature of some problems faced by this field. It introduces the Harvard New Pathway, the larger educational experiment of which this project is a part. It explains the role played by information technology, in general, and by knowledge based physiologic modeling, in particular, in the New Pathway and how they support its overall goals.

1.1. Medical Education: Past and Present

The system of undergraduate[2] education currently in use at most North American medical schools is the product of an ongoing evolutionary process dating back to the Middle Ages [1]. This evolution has often been discrete, rather than continuous, and the present character of medical education has remained virtually unchanged since the beginning of this century . In contrast to this stasis of medical pedagogic technique, the character of medical practice and the scope of medical knowledge have changed drastically over the same period of time. The resulting asynchrony has been the source of important problems.

Eighty years ago, the goal of medical education was to prepare the majority of students for solo general practice [2]. It could, and did, reasonably accomplish this goal by imparting to medical students a circumscribed body of knowledge (the *Basic Science* curriculum[3]) and by engaging them in the intensive apprenticeship experience of clinical clerkships and internship. The graduates of such an education were well prepared for competent practice throughout their professional lifetimes, as the knowledge which they had learned remained largely valid for that period.

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[2] *Premedical* education refers to studies leading to a baccalaureate level degree before entering medical school. *Undergraduate* medical education is the three or four years of medical school prior to obtaining an M.D. The internship, residency, and fellowship training which follows is usually called *postgraduate* medical education.

[3] The principles of structure and function of the human body in health and disease, as conveyed by the sciences of Anatomy, Histology, Biochemistry, Physiology, Pathology, Pharmacology, etc. The systematic introduction of the Basic Sciences into North American medical education was the most recent quantum change in this field. It was inspired by the now famous Flexner Report [3], published in 1910.

The volume of medical knowledge is now a thousand times as large as it was in 1900 and, like other scientific knowledge, it continues to grow at an exponential rate [4]. Yet traditional undergraduate medical education remains committed to imparting the mass of basic and clinical sciences to its students, greatly exceeding their capacity to incorporate this knowledge in a very limited time span (usually the first two years of medical school). Even if medical students were to somehow absorb all this material, it would not serve them well, as large areas are certain to obsolesce rapidly. Thus a system of undergraduate education designed to impart professional knowledge of all medical science, which will last a lifetime, is no longer feasible.

Today, most physicians pursue specialty and subspecialty training after receiving their M.D. degrees[4] [2]. An alternative view of medical learning, which is consonant with this present day reality, regards the undergraduate years as a period of general professional education, the initiation to a lifelong process of learning and development which is a prerequisite for competent clinical practice. This view has long been advocated by members of the education community [5, 6], but has not had a significant impact on medical school curricula. More recently, the Association of American Medical Colleges has called for a major reform of undergraduate medical education along precisely these lines in the GPEP Report[5] [7].

If traditional medical education has become ineffectual by virtue of its emphasis on rapidly imparting a huge body of knowledge, what does the approach of general professional education offer as an alternative? Simply stated, the alternatives to direct knowledge acquisition are *attitudes* and *skills* which will encourage and equip physicians to maintain a lifelong learning process, in order to cope with the explosive growth of medical knowledge. A fundamental requirement of such lifelong self-education is an *active* role on the part of the learner. This too is in contrast to standard medical pedagogic practice, whereby students are the object of countless hours of lectures, demonstrations, and audio-

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[4]Note that this too is (at least in part) a consequence of the volume burden and rapid growth rate of medical science, as physicians strive to attain comprehensive knowledge of at least one small domain. This trend toward increasingly narrow specialization has already caused severe fragmentation of the process of medical care and, if unchecked, is likely to further accelerate this phenomenon in the future.

[5]Published 74 years after the Flexner Report, the GPEP Report is expected to be a harbinger of equally great change. However it is much too soon to assess whether such change has indeed begun to occur.

visual or computer based tutorials which they are expected to incorporate, integrate, and regurgitate in rapid succession.

1.2. The Harvard New Pathway

Motivated by the issues discussed above, Harvard Medical School has initiated an experimental undergraduate medical education program, the *New Pathway* [8]. The developers of the New Pathway have proposed a specific set of attitudes, skills, and knowledge which would prepare students for lifelong professional learning. The principal categories of these are shown in Table 1-1. The goal of the New Pathway curriculum is to provide an environment where the student can acquire these attitudes, skills, and knowledge.

The detailed structure of the New Pathway curriculum and the criteria by which it will be evaluated are beyond the scope of this thesis. However, it is pertinent to note that the New Pathway is a major undertaking, drawing resources from diverse sectors of the Harvard educational community. The curriculum has been under development since July 1982 by a faculty of approximately 30 persons, with extensive input from medical students.

In September 1985, the first New Pathway group, consisting of 24 of Harvard's 165 member first year medical class, was selected[6]. This group, known as the Oliver Wendell Holmes Society, has its own lectures, tutorials, and laboratory exercises and the course material is studied by system rather than discipline[7]. Clinical exposure begins in this first year, and cases are the motivation for and focus of Basic Science learning. The 24 students are divided into four tutorial groups and there is a strong emphasis on teamwork and cooperative learning within these. To date, the New Pathway students, faculty, and staff have been enthusiastic about their experimental program and appear to have developed a very good rapport with each other.

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[6]Each newly accepted medical student was given the opportunity to volunteer for the New Pathway. There were 70 volunteers from among the 165 students, and of these, 24 were selected by the New Pathway faculty.

[7]For example, the anatomy, histology, biochemistry, and physiology of the cardiovascular system are studied as a single block rather than being divided among sections of four different courses.

1. Attitudes:
    a. Attitudes toward patients and colleagues.
    b. Attitudes toward society at large.
    c. Attitudes toward learning.
    d. Attitudes toward one's self.
2. Skills:
    a. Acquiring information from and about patients.
    b. Obtaining, retrieving and storing information.
    c. Working effectively with one's peers and the health care team.
    d. Communicating effectively with patients, families, and colleagues.
    e. Performing basic diagnostic and therapeutic procedures.
    f. Problem solving.
3. Knowledge:
    a. An understanding of the patient as a living being.
    b. An understanding of the patient as an individual and social being.
    c. An understanding of the principles of prevention and of therapeutic strategies.
    d. An understanding of the statistical and probabilistic aspects of human biology and clinical medicine.
    e. An understanding of the complex texture of knowledge and the importance of detail.

Table 1-1: The New Pathway: Foundations of Lifelong Professional Competence

1.3 The Role of Information Technology

One aspect of the New Pathway's approach to medical education is a fundamental commitment to exploit information technology [illegible] and faculty member has a personal computer workstation connected to a comprehensive network and a central database. This system is used [illegible] computer based educational programs which have been specifically developed [illegible] and also to support such routine information management activities as text processing and electronic mail.

There are several important reasons for adopting a prominent role to information technology within this educational environment. [illegible] is a potentially powerful tool to help physicians [illegible]. The individual professional workstation, combined with [illegible] can become a keystone in the lifelong independent learning process [illegible] refer to footer. The early introduction of computers and [illegible] the medical curriculum is anticipated to forestall the reluctance which [illegible] has shown toward the use of [illegible].

The development is being [illegible] the New Pathway [illegible] Faculty, [illegible] some (eg. Module 6, Clinical Medicine [illegible]) computer science research before becoming [illegible] the various modules, the intent is to create [illegible] common format of [illegible] elements of patient [illegible] may be initially applied in the implementing construction of Module 5, Simulation of Biological Phenomena, with additional relevance to several other categories listed in Table 1-2. Specifically, my goal was to build an intelligent [illegible] supporting the [illegible] This system was to be intelligent by [illegible] human physiology. Its value as a pedagogic tool was to be a consequence of its ability to describe the nature of physiologic [illegible] to explain the [illegible] was expected to [illegible]

1. Vocabulary Building
2. Computer-Stored Course Notes
3. Bibliographic Reference Files
4. Access to Data Bases
5. Simulation of Biological Phenomena
6. Clinical Problem Solving Applications
7. Computer-Based Medical Records
8. Computer-Based Test Bank Questions
9. Personal Reference Files

Table 1-2: Proposed Software Modules for the New Pathway

The development is being carried out by teams composed of members of the New Pathway faculty, members of the instructional technology and programming staff, post-doctoral fellows, and medical students taking elective rotations in educational technology. It is envisioned that the programs will be developed, evaluated, and modified over several years of student use.

1.4. Intelligent Physiologic Modeling

The hypothesis which motivated this project is that certain elements of artificial intelligence and knowledge based systems technology might be fruitfully applied in the construction of Module 5, Simulation of Biological Phenomena, with additional relevance to several other categories listed in Table 1-2. Specifically, my goal was to build an *intelligent physiologic modeling system* for use in this module. This system was to be intelligent by virtue of domain specific knowledge which was encoded into models of different aspects of human physiology. Its value as a pedagogic tool was to be a consequence of its ability to describe the nature of physiologic entities, to explain the relationships between them, and to ascribe causality to their interactions, in addition to numerical and qualitative simulation of

their dynamic behavior. The system was intended to provide support for several distinct physiologic models which would necessarily overlap in content. This approach corresponds to clinical physiologic reasoning and to the way in which physiology is currently taught to medical students. It does not represent a unified description of all bodily functions.

This project has consisted of the design, implementation, and preliminary evaluation of a Knowledge Based Physiologic Modeling System (KBPMS) arising out of the above desiderata. The project has had a truly symbiotic relationship with the New Pathway of which it represents but a very small part. KBPMS provided the New Pathway with an interesting pedagogic tool, based on developments in artificial intelligence research. The New Pathway, in turn, provided an opportunity for evaluation and refinement of KBPMS and the computational techniques upon which it is based.

2. Background and Related Work

The development of KBPMS has been based on previous work in two major fields of endeavor: computer applications to education, and computer based physiologic modeling. This chapter reviews some of the work in these fields and describes their relationship to this project.

2.1. Computer Applications to Education

Computer assisted instruction (CAI) has been available as an educational tool for over 25 years. Traditional CAI is an adaptation of the programmed learning approach whereby a flowchart guides the student through progressively more difficult subject matter. Multiple choice questions are used to assess the students' progress, and in response to his answers, material may be reviewed, or the pace of the presentation slowed down or accelerated. The student's responses are also available for evaluation purposes.

A number of variations on the basic theme of CAI have taken place in several settings. The PLATO system [11] was the first to incorporate graphics capabilities by using a plasma display and microfiche projector. More recently, slide-tape, videotape, and videodisk interfaces have been developed for the same purpose. The increasing availability of home computers has greatly facilitated the dissemination of CAI material directed toward a wide range of age and interest groups.

Much effort has been invested in applying CAI techniques to medical education [12], but the resulting programs have not been widely integrated as routine parts of medical curricula. Some medical educators feel that this lack of success has been due to centralization of such systems around large mainframe computers, and to inadequate user interfaces [13]. They suggest that the advent of high quality audiovisual interfaces and low cost microcomputer based systems will revitalize medical CAI. Yet traditional CAI systems, in medicine as well as other areas, are limited in a more fundamental way. Their flowchart foundations render them inflexible, unable to adequately adapt to the needs of individual students. Viewed from the perspective of the New Pathway program, they do not encourage a sufficiently active role for the student in the learning process.

In response to the deficiencies of traditional CAI, several investigators have attempted to apply artificial intelligence technology to the task of producing intelligent tutoring

systems [14]. Such systems, termed intelligent CAI (ICAI), attempt to be intelligent in two different ways. First, they incorporate comprehensive knowledge of the domain they are intended to teach, and second, they contain a model of student behavior and modify their tutelage in response to what the student seems to understand or misunderstand. To date, most ICAI systems have been applied in highly structured domains such as game playing [15] and troubleshooting electronic circuits [16], where they have met with preliminary success.

GUIDON [17, 18] is an ICAI program designed to teach the knowledge encoded in rule based "expert" systems. An initial version uses the MYCIN infectious disease knowledge base [19] and thus represents the first application of ICAI to medical education. One of the important lessons learned during the development of GUIDON was that a set of rules which provides good consultation performance may none-the-less be awkward for use by such a pedagogic program. Thus the developers of GUIDON have undertaken the task of restructuring the MYCIN knowledge base to make it suitable for teaching purposes. This is the NEOMYCIN project [20].

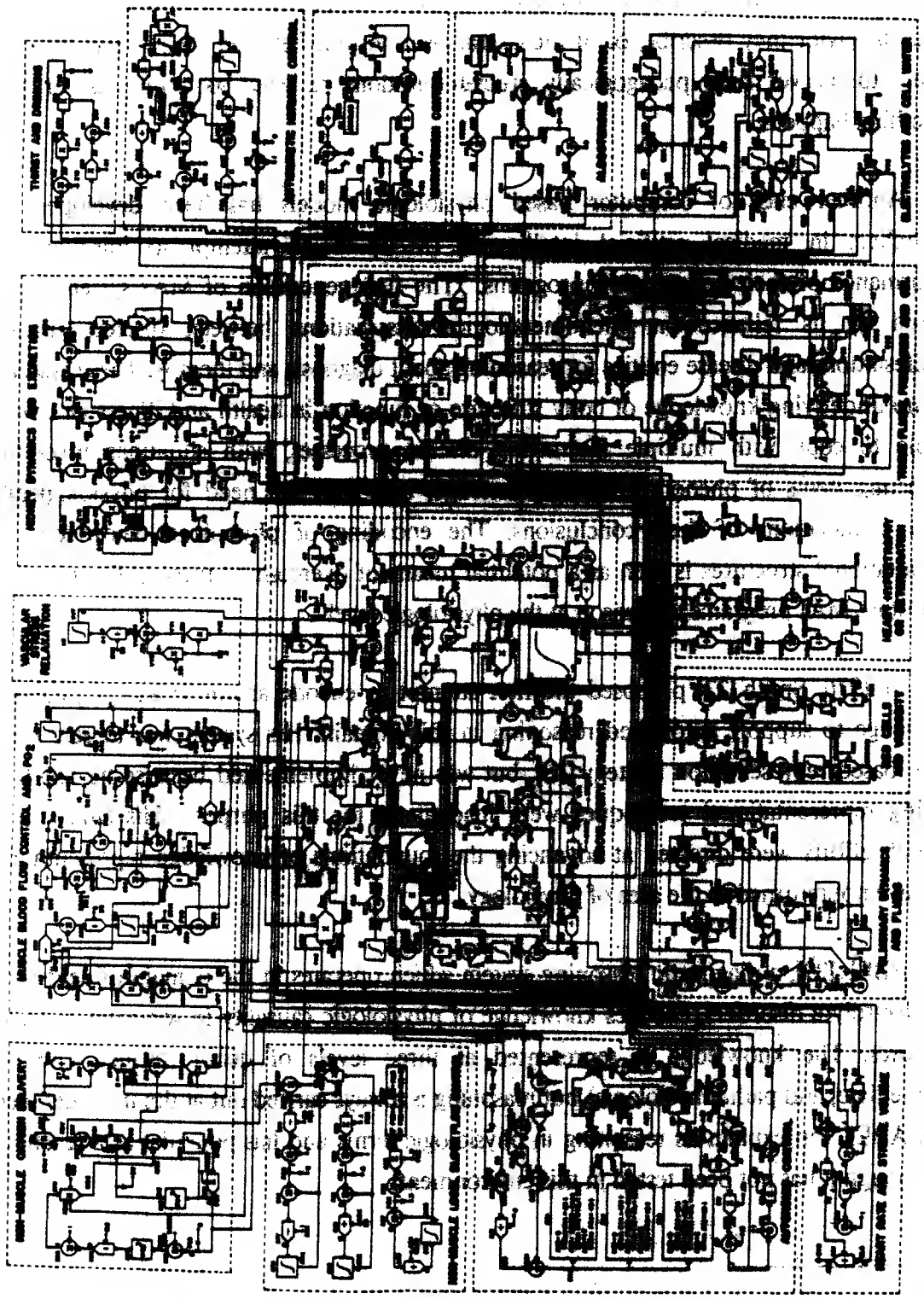
KBPMS occupies a middle ground between traditional CAI and the more advanced ICAI systems. It has deep knowledge of its domain, but does not attempt to model the student's behavior or level of comprehension. It is intended to provide a flexible educational tool based on well established pedagogic and computational principles, but does not address entirely new research issues in either field.

2.2. Computer Based Physiologic Modeling

The earliest computer models of physiologic function were numerical implementations of mathematical relationships which had been empirically determined to exist among physiologic variables. Some of these numeric models have become quite elaborate, describing large areas of human physiology and pathophysiology. Guyton has developed such a model of the entire cardiovascular system [21] and Dickinson has built the equivalent for respiratory physiology [22]. Figure 2-1 is a schematic representation of HUMAN [23], a very large numerical model which is used as a medical teaching aid.

Numeric physiologic models are analogous to the spreadsheet programs which have become popular in the business community. They allow the user to observe the response to

Figure 2-1: HUMAN: A Numerical Model of Physiology



a given set of perturbations and are amenable to graphic presentation of results. However, like spreadsheet programs, they are limited by their depth of knowledge in the domain which they are modeling. Specifically, they have no knowledge of the nature of the entities whose behavior they describe, or of the causal relationships between them. Thus they are unable to answer such pedagogically crucial questions as *what* something is, or *why* something happens.

Another class of computer based physiologic models has been developed by researchers in medical artificial intelligence (MAI) in an attempt to improve the performance of their consultation programs. The first generation of MAI systems[9] was limited by its reliance on phenomenological associations between discrete disease manifestations and disease entities for reasoning about diagnosis and therapy. These systems had no underlying knowledge of body structure or function in health and illness and were unable to cope with multiple interacting disease processes, with situations involving conflicting items of phenomenologic knowledge, and with the need for physiologically based explanations of their conclusions. The encoding of physiologic knowledge in computational structures is seen as a potential means of overcoming these deficiencies by permitting MAI programs to "reason at the physiologic level".

In 1976, Smith [27] proposed the first attempt to encode anatomic and physiologic knowledge to support automated reasoning in this domain. His system was to rely on a frame based representation strategy [28], but was never implemented because he felt that existing representational techniques were inadequate for this purpose. His subsequent research efforts were directed at advancing the foundations of knowledge representation and have not returned to the area of physiology.

ABEL [29], a diagnostic reasoning system which operates in the domain of acid-base and electrolyte disorders, encodes knowledge of physiologic causality in semantic network structures. The knowledge is represented at three levels of detail (termed clinical, intermediate, and pathophysiologic) but reasoning is always carried out at the most detailed level. ABEL can explain its reasoning in physiologic terms and has potential as a teaching tool, though it has not been tested in this environment.

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[9] The ubiquitous "big four": MYCIN [19], INTERNIST-1 [24], CASNET [25], and PIP [26].

Several investigators are currently exploring the encoding of knowledge of various medical domains in different types of causal networks. Wallis and Shortliffe [30] have combined causal and rule based representations in a prototype medical explanation production system. Long [31] has developed a causal physiologic model of cardiovascular disease for use in diagnostic and therapeutic reasoning about angina and heart failure. Blum [32] has devised a system for representation of empirically derived causal relationships as a means of extraction of knowledge from a large clinical database. Kunz [33] has studied the analysis of physiologic models for encoding in knowledge based systems. Widman [34] has developed a representation method for dynamic causal knowledge in the domain of cardiovascular physiology. All these efforts have, in turn, relied on more foundational work on the representation and simulation of causality in physical mechanisms, such as that carried out by Rieger and Grinberg [35], deKleer [36, 37], and others.

A similar, yet distinct, approach to physiologic modeling has developed as a consequence of recent advances in qualitative process theory [38]. Qualitative simulation [39] models a physiologic system by propagating constraints imposed by the mathematical relationships among physiologic variables. The numeric values of these variables need not be explicitly known, but may be characterized in terms of their direction and rate of change, as well as their magnitude relative to pertinent landmarks (eg. above or below normal). Given an initial state and perturbations, qualitative simulation can determine all mathematically possible successor states, though some of these may not reflect physically possible behaviors of the system being modeled.

NEPHROS [40] combines the ideas of causal physiologic representation and qualitative physiologic simulation. It is a modeling system based on the propagation of constraints through a series of *gray boxes* representing functional units of the human body. The gray boxes may be either discrete entities or further decomposable into structures made up of other gray boxes, thus implementing a hierarchic representation based on level of descriptive detail. NEPHROS has a simple model of renal function and is capable of reasoning about the pathophysiologic entities of congestive heart failure, the syndrome of inappropriate antidiuretic hormone secretion, and the nephrotic syndrome.

KBPMS occupies an intermediate position in this spectrum of modeling methodologies, analogous to its stance relative to other computer based education aids. It incorporates the principles of numeric and qualitative simulation as well as the

representation of causal relationships at varying levels of detail. It is capable of reasoning about *what* something is and *why* something happens. It emphasizes the application of these features as educational tools, over their further theoretical enhancement.

3. Project Overview

This chapter briefly summarizes the design, implementation, and evaluation phases of the KBPMS project. A more detailed description of each is then presented in Chapters 4 and 5.

3.1. Design

The design phase of this project consisted of the development of an intelligent physiologic modeling system (KBPMS) with the following capabilities:

1. *Compilation.* KBPMS is able to compile[10] models encoding different areas of physiologic knowledge into internal computational structures for use in carrying out its functions. The models are written in a uniform frame based language [28], independent of the computational structures onto which they are mapped, and independent of the procedural mechanisms by which simulation and explanation are carried out.

2. *Simulation.* KBPMS accepts perturbations to a model in any of the following forms: numerical values of parameters, qualitative characterizations of parameters, or impairment of processes. It shows the resulting numerical and qualitative perturbations as they propagate through the model. Following a simulation run, KBPMS can explain why a particular event occurred.

3. *Explanation.* KBPMS is able to answer the following questions, where the blanks may be filled by any entity which is described in a model:

   What is _____?
   What directly influences _____?
   What is directly influenced by _____?
   Does _____ influence _____?
   What are the mechanisms of _____?

4. *Verification.* When compiling a model, KBPMS performs limited checking to ensure internal consistency. The definition of this consistency is given in terms of details of model structure in Section 4.2 (page 20).

[10] Though not necessarily combine.

3.2. Implementation

The implementation phase of this project was twofold. Initially, a prototype was constructed in LISP [41] for a mainframe system at the MIT Laboratory for Computer Science. The prototype implemented most features of the design described above, and was fully debugged and operational using a simple model of respiratory physiology. It had a sufficiently powerful user interface to permit extensive testing, but lacked any graphics or advanced user support capabilities.

A subsequent implementation was constructed in MUMPS [10] using an HP-150 microcomputer provided by the Harvard New Pathway. This final implementation possesses all the specified design capabilities in addition to an advanced, friendly, user interface. This interface features extensive use of interactive graphics and is fully compatible with other elements of the New Pathway educational software. The final version uses the same respiratory model as the prototype.

3.3. Evaluation

Ideally, the evaluation objective of this project would have been to fully assess the long term effectiveness of KBPMS as an educational resource within the context of the New Pathway. However, such conclusive and rigorous evaluation was unrealistically ambitious for the limited time span of the project (approximately one year). Therefore an attempt was made at some form of limited but potentially reproducible outcome measurement, with the prior realization that the results could be considered "soft".

The evaluation phase consisted of a homework exercise in respiratory physiology which was given to all New Pathway students using standard educational resources (class notes, textbooks, laboratory materials, etc.). In addition, a randomly selected half of the students also had access to KBPMS while performing this homework. A brief evaluation quiz was subsequently given to all the students. The remainder of the students were then given access to KBPMS. All students and faculty were encouraged to report their opinions of KBPMS, its major strengths and weaknesses. All participation in this evaluation experiment was solicited on an entirely voluntary basis and one third of the 24 students complied.

Analysis of the quiz scores of the two groups indicated that those students who had

access to KBPMS scored approximately the same as those who had not. The mean scores were 78% and 76% respectively with a standard deviation of 27%. Due to this very small difference and the small sample size (n=8), these results are not statistically significant. Student and faculty evaluation of KBPMS spanned a broad spectrum of opinions. These ranged from those who thought the program and respiratory model were exceedingly simplistic to those who felt they were far too complex, with a variety of comments inbetween and no general consensus.

4. Methods

This chapter describes the methods used in carrying out the KBPMS project. It presents definitions of the relevant physiologic concepts, the knowledge representation scheme which was employed, the algorithms for carrying out the modeling system's functions, and details of the evaluation strategy. These are illustrated with examples drawn from respiratory physiology.

4.1. Definitions

The methods described below presuppose a highly simplified view of human physiology, one which may be fully described in terms of *parameters*, *processes*, *states* and *steps*. For this purpose the following definitions apply:

- A *parameter* is any potentially measurable physiologic entity. It may have any simple or combined units and thus may be an amount, concentration, rate, etc. (eg. tidal volume, respiratory rate).

- A *process* is a description of the way in which parameters interact. For example, the process of bulk gas flow describes the interaction of the parameters: respiratory rate, tidal volume, dead space, and alveolar ventilation. Increased respiratory rate increases alveolar ventilation, increased dead space decreases alveolar ventilation, and so forth.

- A *state* is a characterization of the qualitative value of a parameter (increased, decreased, or normal) or the status (active or impaired) of a process. For example, hyperventilation is a state characterized by decreased arterial pCO_2 (a parameter), and adult respiratory distress syndrome is a state associated with impaired alveolocapillary diffusion (a process).

- A *step* of a physiologic simulation is the set of changes of values of parameters indicated by the relationships described by a single process. Thus the following would each describe single steps: "Alveolar ventilation (a parameter) is increased by increased respiratory rate (a parameter) through bulk gas flow (a process)."; "CO_2 excretion rate (a parameter) is decreased and arterial pCO_2 (a parameter) is increased by increased alveolar pCO_2 (a parameter) through alveolocapillary diffusion (a process)."

4.2. Knowledge Representation

Figure 4-1 describes the relationship between two parameters: metabolic CO_2 production rate (CO_2 prod) and CO_2 excretion rate (VCO_2). In this figure, and all KBPMS diagrams, parameters are represented by squares and processes are represented by triangles. The influence of one parameter on another is shown using solid lines with arrowheads indicating the direction of the influence being mediated. Thus CO_2 production influences VCO_2 through CO_2 elimination (CO_2 elim).

Figure 4-1: An Abstract View of CO_2 Elimination

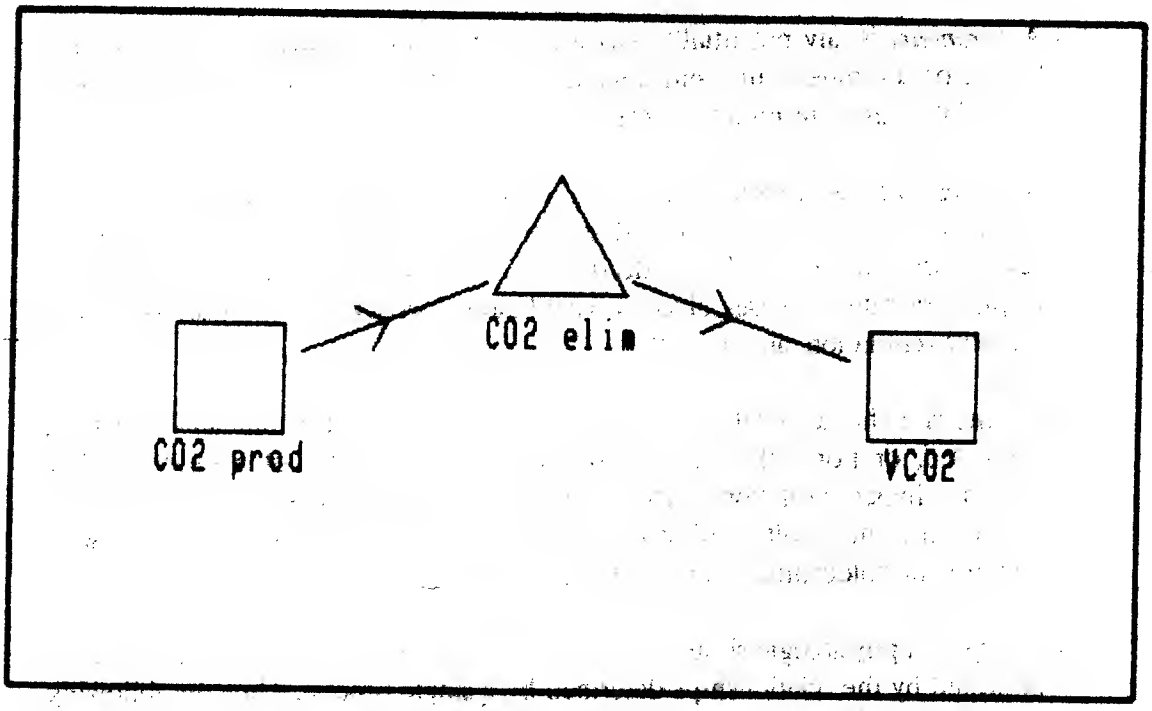
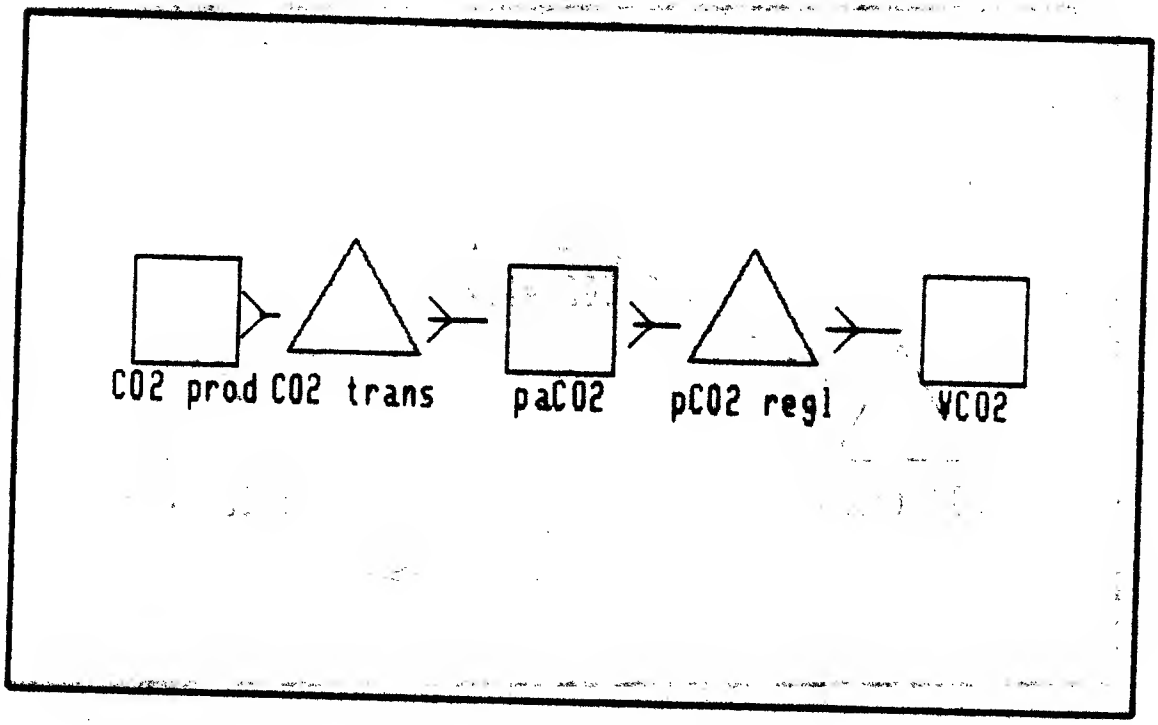


Figure 4-2 shows the same relationship at a greater level of detail. CO_2 production influences arterial pCO_2 ($paCO_2$)[11] through circulatory CO_2 transport (CO_2 trans) and paCO2 influences VCO_2 through respiratory regulation of $paCO_2$ (pCO_2 regl), in series. The overall influence of CO_2 production on VCO_2 must, of course, be consistent at all levels of detail, but Figure 4-2 encodes more fine grained knowledge than Figure 4-1.

Figure 4-2: A More Detailed View of CO_2 Elimination



[11] pCO_2 is the partial pressure of carbon dioxide gas. Unless otherwise specified, it refers to the partial pressure of CO_2 dissolved in arterial blood, also designated as arterial pCO_2 and $paCO_2$. Alveolar pCO_2 ($pACO_2$) is the partial pressure of CO_2 in alveolar gas. Unfortunately, this widely accepted usage of abbreviations can be somewhat confusing.

Circulatory CO_2 transport and respiratory regulation of $paCO_2$ are the *mechanisms* of CO_2 elimination as shown in Figure 4-3. These processes may, in turn, have mechanisms which we wish to similarly describe, suggesting a strict taxonomy such as shown in Figure 4-10 (page 33). Here, as in all other KBPMS diagrams, the taxonomic links are indicated by dashed lines.

Figure 4-3: The Mechanisms of CO_2 Elimination

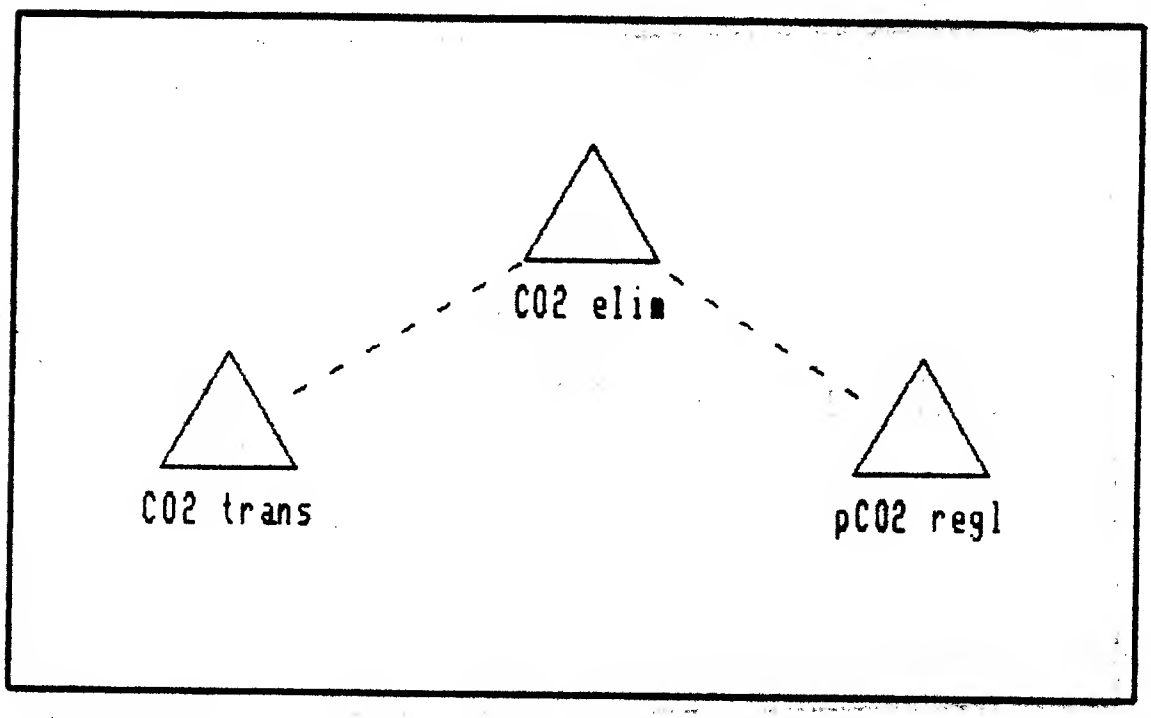
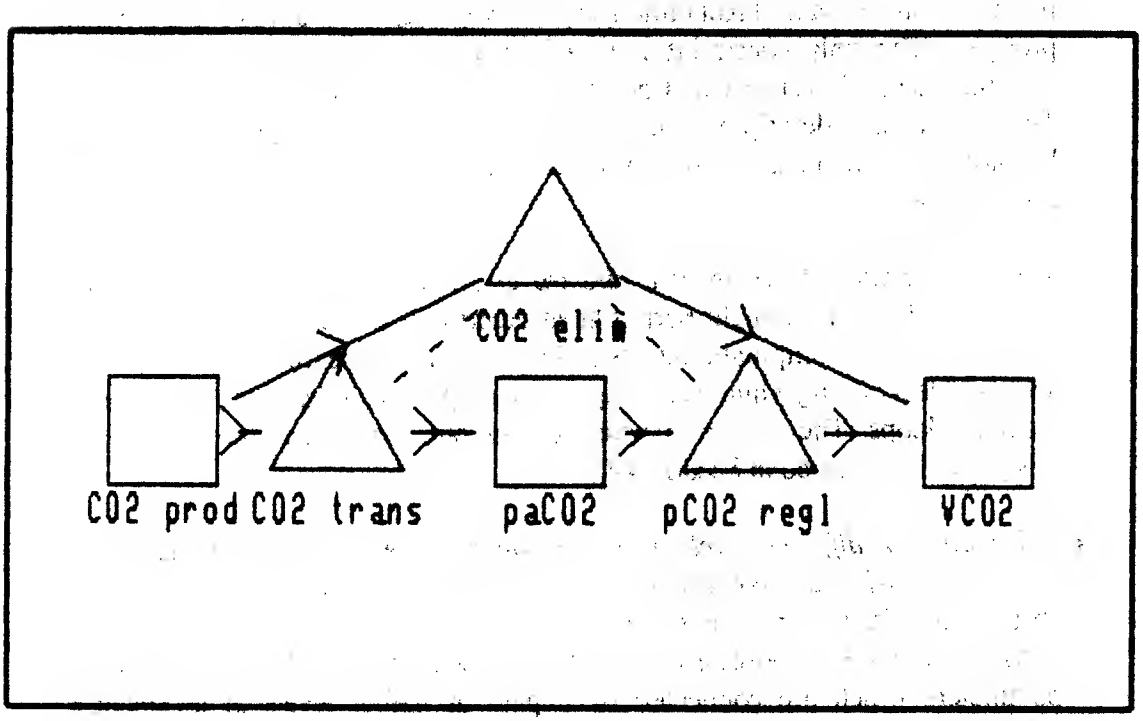


Figure 4-4 is a unified representation of the preceding three figures and shows influence as well as taxonomic links. Note that $paCO_2$ has no meaning in terms of the process of CO_2 elimination, and is only meaningful at the second taxonomic level.

Figure 4-4: A Unified Representation of CO_2 Elimination



The following are some properties of the representation strategy illustrated above. The examples are taken from Figures 4-8, 4-9, and 4-10 (pages 31 to 33).

- *Parameters can be very different things.* They are united only by the property that they all have a potentially measurable value. For example, respiratory rate is routinely measurable and clinically important while alveolocapillary diffusion rate is somewhat esoteric and quite difficult to measure, yet both are parameters.

- *Processes can be very different things.* They are united only by the property that they describe the interaction of parameters. CO_2 elimination is a very abstract concept while bulk gas flow is a very specific physical phenomenon, yet both are processes.

- *Processes at the same taxonomic level need not have the same descriptive level of abstraction.* In Figure 4-10 pulmonary gas transport, which is a physiologic process, is at the same taxonomic level as alveolocapillary diffusion, a physical process. The only constraint imposed by the taxonomy is that a process' children be at a lower or equal descriptive level of abstraction than the parent. The pertinent descriptive levels are: pathophysiologic, physiologic, biochemical, chemical, and physical. The *summum genus* process is homeostasis.

- *The representation structure is a directed graph in which cycles are permitted.* This type of structure might permit great descriptive power, though it may also incur substantial computational cost. At the very least, care must be taken to avoid endless cycling while traversing such a graph which describes one or more feedback loops. The algorithm which carries out simulation and circumvents this pitfall is described in Section 4.4.

- *Information at different levels of abstraction must be consistent.* A necessary, though not sufficient, condition for consistency is that at any given level of abstraction (defined by the taxonomic, rather than the descriptive, level of abstraction of the processes involved) there must exist a path between every two parameters which are connected by a path at higher levels of abstraction. Intuitively, a more detailed description must explain at least as much as a more abstract one.

Parameters, processes, and states are not discrete entities, rather they all have an internal structure which may be described using frames. A parameter frame describing pCO_2 is shown in Figure 4-5[12]. The type, names, descriptions, references, units, clinical-measurability, normal-range, and physiologic-range slots contain the indicated static information. The associated-states, influencing-processes, and processes-influenced[13] slots are filled from information in the state frames and process frames at the time that a model is compiled. In the the latter two slots, the lists of processes are sorted by their taxonomic level. The default-qualitative-value and default-numeric-value slots are used to initialize parameter values, which are then dynamically altered during simulation. The following qualitative values are permitted: normal, increased, decreased, further increased, further decreased, increased toward normal, decreased toward normal, and unknown. These are discussed in Section 4.3 along with the qualitative operators used to manipulate them.

---

[12]Figures 4-5, 4-6, and 4-7 show both the LISP and MUMPS versions of the corresponding frames. In MUMPS, strings are used as indexes to globals (multidimensional arrays) and therefore "↑global(model,frame,slot,index)=value" represents the corresponding LISP frame construct. The Figures show a formatted version of the MUMPS frames which include two slots not found in their LISP counterparts. These are icon and glab which are used for the graphics component of the MUMPS version of KBPMS.

[13]Unfortunately, *influence* is not a very good choice of words here. Parameters really only influence other parameters through processes. The names of these slots seem to imply that parameters also influence processes *per se* and vice-versa. Strictly speaking, this is not the case.

Figure 4-5: A Parameter Frame

```
(PCO2
     (type: (PARAMETER))
     (names: ("arterial pCO2" "pCO2" "paCO2"))
     (description: ("partial pressure of carbon dioxide in arterial blood"))
     (references: "West p. 1")
     (units: ("mm Hg"))
     (clinical-measurability: (ROUTINE))
     (normal-range: (38.0 42.0))
     (physiologic-range: (16.0 100.0))
     (associated-states: (HYPERCAPNEA NIL HYPOCAPNEA))
     (influencing-processes: ((2 (CIRCULATORY-CO2-TRANSPORT RESPIRATORY-CONTRO
                                  L-OF-PCO2))
                              (3 (CIRCULATORY-FLOW PULMONARY-GAS-EXCHANGE))
                              (4 (ALVEOLOCAPILLARY-DIFFUSION))))
     (processes-influenced: ((2 (RESPIRATORY-CONTROL-OF-PCO2))
                             (3 (CHEMOREFLEXES))))
     (default-qualitative-value: (NORMAL))
     (default-numeric-value: (40.0)))


PCO2
----

     ASSOCIATED-STATES:    HYPERCAPNEA
                           NIL
                           HYPOCAPNEA
     CLINICAL-MEASURABILITY:    routine
     DEFAULT-NUMERIC-VALUE:    40
     DEFAULT-QUALITATIVE-VALUE:    normal
     DESCRIPTION:    partial pressure of carbon dioxide in arterial blood
     GLAB:    paCO2
     ICON:    .476
              .666666666667
              1
              4
              46
     INFLUENCING-PROCESSES:    CIRCULATORY-CO2-TRANSPORT,RESPIRATORY-CONTROL-OF
                               -PCO2
                               CIRCULATORY-FLOW,PULMONARY-GAS-EXCHANGE
                               ALVEOLOCAPILLARY-DIFFUSION
     NAMES:    arterial pCO2
               pCO2
               paCO2
     NORMAL-RANGE:    38
                      42
     PHYSIOLOGIC-RANGE:    16
                           100
     PROCESSES-INFLUENCED:    RESPIRATORY-CONTROL-OF-PCO2
                              CHEMOREFLEXES
     REFERENCES:    West p. 1
     TYPE:    parameter
     UNITS:    mm Hg
```

Figure 4-6 shows a process frame describing bulk gas flow. The type, names, description, references, teleology, and descriptive-level-of-abstraction slots contain the corresponding items of static information. The influencing-parameters and parameters-influenced slots[14] contain incoming and outgoing influence links respectively. The is-a-mechanism-of and mechanisms slots contain upward and downward taxonomic links respectively. (The mechanisms slot of the bulk gas flow frame contains NIL because the mechanisms of bulk gas flow are not described in this model.) The quantitative-rules slot, contains an arithmetic expression composed of influencing-parameters, constants, and a parameter from the parameters-influenced list. The qualitative-rules slot contains an influencing-parameter, qualitative algebraic operators, and influenced-parameters, respectively. The operators may be one of the following: monotonically increasing (M+) or monotonically decreasing (M-). The semantics of these operators is discussed in the next section. The default-status slot indicates whether this process is normally active or impaired.

[14] Also misnomers in the same sense as previously indicated.

Figure 4-6: A Process Frame

```
(BULK-GAS-FLOW
     (type: (PROCESS))
     (names: ("bulk gas flow"))
     (description: ("flow of gas volumes through the tracheobrochial tree"))
     (references: "Guyton pp. 484-486" "West pp. 15-19")
     (teleology: NIL)
     (descriptive-level-of-abstraction: (PHYSICAL))
     (associated-states: (COPD))
     (influencing-parameters: (RESPIRATORY-RATE TIDAL-VOLUME DEAD-SPACE))
     (parameters-influenced: (ALVEOLAR-VENTILATION))
     (is-a-mechanism-of: (PULMONARY-GAS-TRANSPORT))
     (mechanisms: NIL)
     (taxonomic-level: (5))
     (quantitative-rules: ((TIMES RESPIRATORY-RATE (DIFFERENCE TIDAL-VOLUME D
                            EAD-SPACE)) ALVEOLAR-VENTILATION)))
     (qualitative-rules: ((RESPIRATORY-RATE (M+ ALVEOLAR-VENTILATION))
                          (TIDAL-VOLUME (M+ ALVEOLAR-VENTILATION))
                          (DEAD-SPACE (M- ALVEOLAR-VENTILATION))))
     (default-status: (ACTIVE)))
```

```
BULK-GAS-FLOW
-------------

     ASSOCIATED-STATES:    COPD
     DEFAULT-STATUS:    active
     DESCRIPTION:    flow of gas volumes through the tracheobrochial tree
     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:    physical
     GLAB:    gas flow
     ICON:    .63125
              .166666666667
              1
              3
              90
     INFLUENCING-PARAMETERS:    RESPIRATORY-RATE
                                TIDAL-VOLUME
                                DEAD-SPACE
     IS-A-MECHANISM-OF:    PULMONARY-GAS-TRANSPORT
     MECHANISMS:    NIL
     NAMES:    bulk gas flow
               gas flow
     PARAMETERS-INFLUENCED:    ALVEOLAR-VENTILATION
     QUALITATIVE-RULES:    RESPIRATORY-RATE->M+:ALVEOLAR-VENTILATION
                           TIDAL-VOLUME->M+:ALVEOLAR-VENTILATION
                           DEAD-SPACE->M-:ALVEOLAR-VENTILATION
     QUANTITATIVE-RULES:    'ALVEOLAR-VENTILATION'='RESPIRATORY-RATE'*('TIDAL-V
                            OLUME'-'DEAD-SPACE')
     REFERENCES:    Guyton pp. 484-486
                    West pp. 15-19
     TAXONOMIC-LEVEL:    5
     TELEOLOGY:    NIL
     TYPE:    process
```

Figure 4-7 shows two state frames describing hypercapnea and adult respiratory distress syndrome, respectively. They are smaller than parameter or process frames and their structure is self-explanatory. Note that state frames also contain a descriptive level of abstraction, and that they may only represent the perturbation of a single parameter or the impairment of a single process. All three types of frames permit multiple synonyms in the names slot and any of their slots may be filled with NIL when the relevant information is inappropriate or unavailable.

Figure 4-7: Two State Frames

```
(HYPERCAPNEA
     (type: (STATE))
     (names: ("hypercapnea" "hypercarbia" "hypoventilation"))
     (descriptive-level-of-abstraction: (PHYSIOLOGIC))
     (perturbation: (INCREASED PCO2)))

HYPERCAPNEA
-----------

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physiologic
     NAMES:    hypercapnea
               hypercarbia
               hypoventilation
     PERTURBATION:    increased
                      PCO2
     TYPE:    state


(ARDS
     (type: (STATE))
     (names: ("ARDS" "adult respiratory distress syndrome" "shock lung"))
     (descriptive-level-of-abstraction: (PATHOPHYSIOLOGIC))
     (perturbation: (IMPAIRED ALVEOLOCAPILLARY-DIFFUSION)))

ARDS
----

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   pathophysiologic
     NAMES:    ARDS
               adult respiratory distress syndrome
               shock lung
     PERTURBATION:    impaired
                      ALVEOLOCAPILLARY-DIFFUSION
     TYPE:    state
```

The examples used above are taken from a model of carbon dioxide homeostasis by the respiratory system. This model was prepared using Guyton's [42] and West's [43] textbooks of physiology. Figure 4-8 illustrates the parameters and processes in this model. The influence links and taxonomic links are shown in Figures 4-9 and 4-10 respectively. Appendix I lists the complete frame based representation of this model. The carbon dioxide homeostasis model is quite small (11 parameters, 11 processes, and 10 states) and encompasses a correspondingly limited subset of medically relevant respiratory physiology. None-the-less, it seems reasonable to assume that a physiologic model encoding the sort of knowledge that we wish to teach medical students about a particular aspect of human physiology (respiratory, cardiovascular, electrolytes, acid-base, neuro, etc.) might be described by models of this order of magnitude (ie. less than 100 parameters and processes).

Figure 4-8: A Model of CO_2 Homeostasis by the Respiratory System

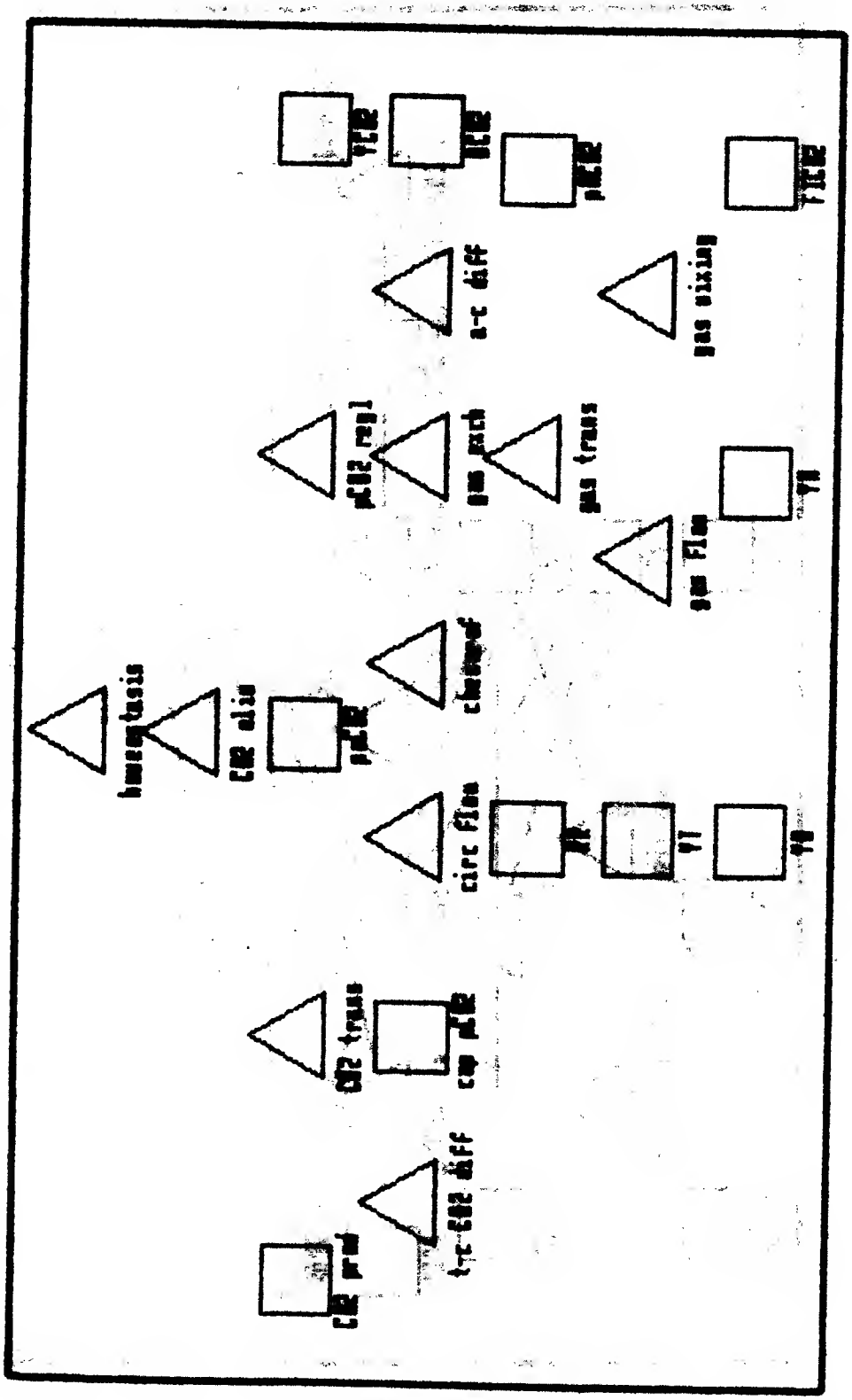


Figure 4-9: The Respiratory Model Showing Influence Links

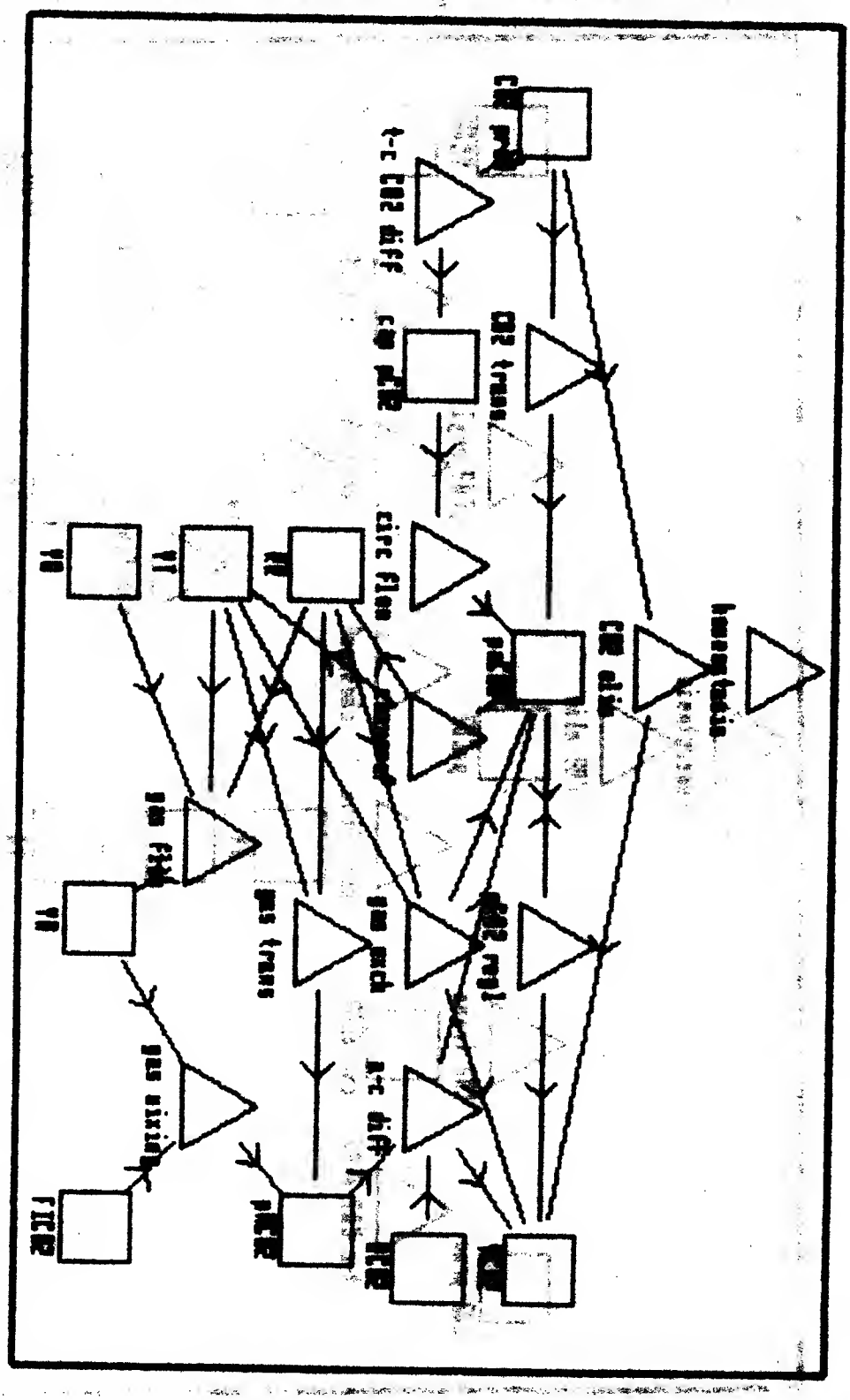
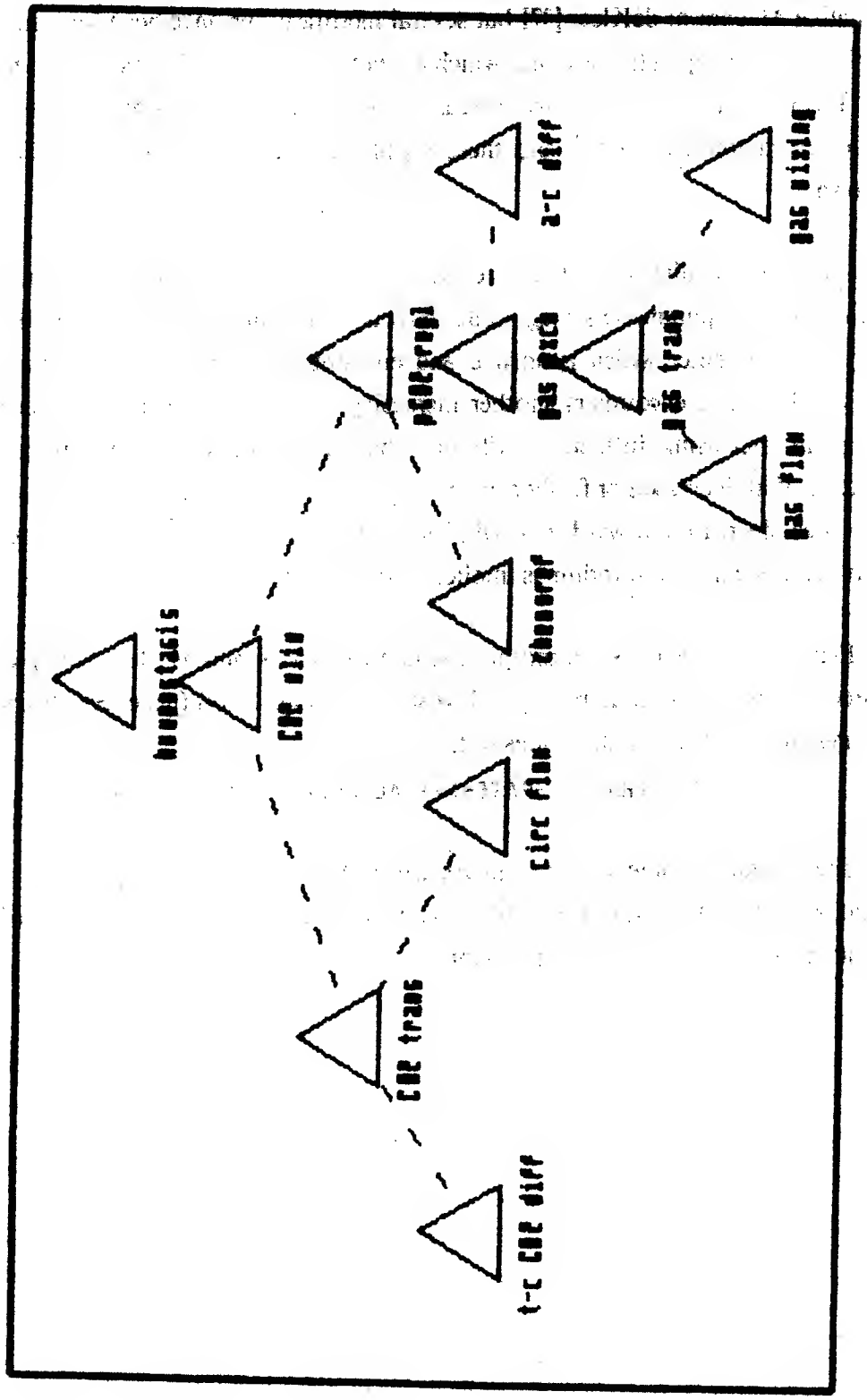


Figure 4-10: The Respiratory Model Showing Taxonomic Links



4.3. Qualitative Values and Operators

The semantics of the qualitative operators M+ and M- are based on the Incremental Qualitative Algebra of deKleer [37] but several modifications have been introduced to deal with the additional qualitative values which are not present in deKleer's original four valued logic. The new values are: further increased, further decreased, increased toward normal, and decreased toward normal; and these supplement: normal, increased, decreased, and unknown.

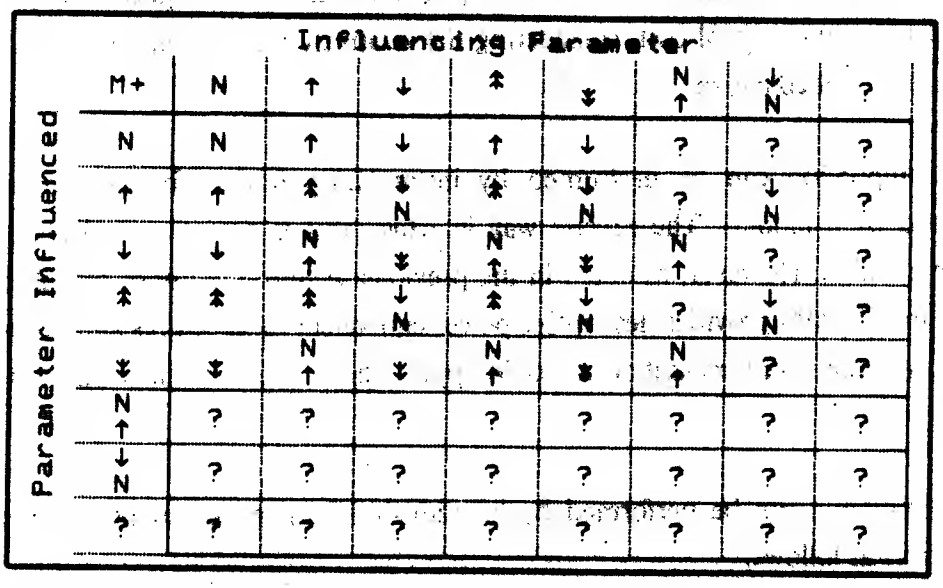
The new qualitative values are somewhat imprecise concepts which encode a combination of a parameter's magnitude, direction and rate of change, as well as its past history. A parameter which is normal and encounters an increasing influence becomes increased. If it then encounters another increasing influence it becomes further increased. Subsequent increasing influences will not change the value of the further increased parameter. If an increased or further increased parameter encounters a decreasing influence it becomes decreased toward normal. The notion of homeostatic forces returning a physiologic system to equilibrium is implicit in this last transition.

Figure 4-11 illustrates the complete semantics of M+ and M-. For example, consider the process frame describing bulk gas flow shown in Figure 4-6 (page 28). It contains the following entry in its qualitative-rules slot:

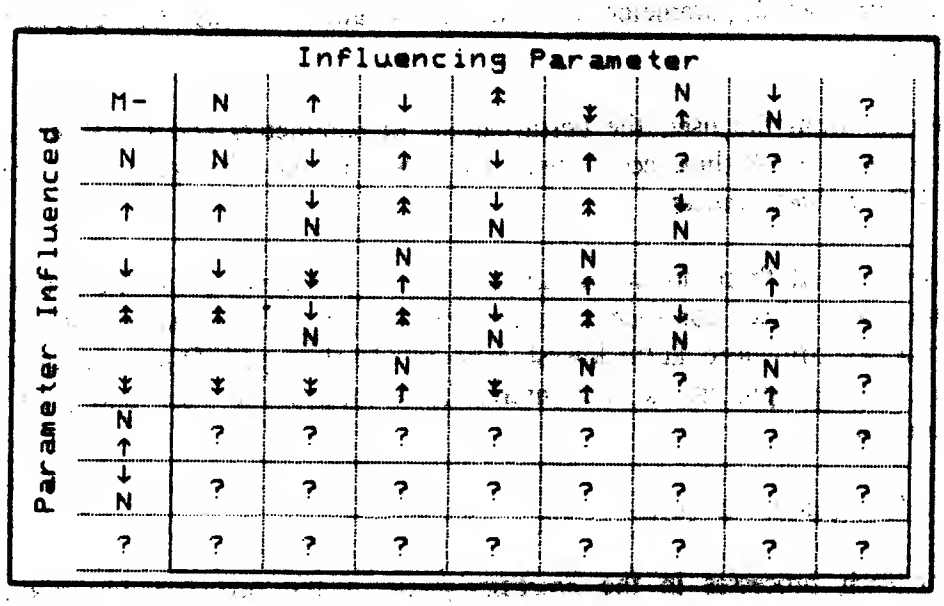
```
RESPIRATORY-RATE->M+:ALVEOLAR-VENTILATION
```

The semantics table for M+ in Figure 4-11 gives the resulting value for alveolar ventilation given its initial value (the parameter influenced) and given the value of respiratory rate (the influencing parameter).

Figure 4-11: Semantics of the Qualitative Operators M+ and M-



Parameter Influenced \ Influencing Parameter: M+	N	↑	↓	↟	↡	N↑	↓N	?
N	N	↑	↓	↑	↓	?	?	?
↑	↑	↟	↓N	↟	↓N	?	↓N	?
↓	↓	N↑	↡	N↑	↡	N↑	?	?
↟	↟	↟	↓N	↟	↓N	?	↓N	?
↡	↡	N↑	↡	N↑	↡	N↑	?	?
N↑	?	?	?	?	?	?	?	?
↓N	?	?	?	?	?	?	?	?
?	?	?	?	?	?	?	?	?



Parameter Influenced \ Influencing Parameter: M-	N	↑	↓	↟	↡	N↑	↓N	?
N	N	↓	↑	↓	↑	?	?	?
↑	↑	↓N	↟	↓N	↟	↓N	?	?
↓	↓	↡	N↑	↡	N↑	?	N↑	?
↟	↟	↓N	↟	↓N	↟	↓N	?	?
↡	↡	↡	N↑	↡	N↑	?	N↑	?
N↑	?	?	?	?	?	?	?	?
↓N	?	?	?	?	?	?	?	?
?	?	?	?	?	?	?	?	?

N - normal

↑ - increased

↓ - decreased

? - unknown

↟ - further increased

↡ - further decreased

N↑ - increased toward normal

↓N - decreased toward normal

4.4. Algorithms

The algorithms required to perform the four tasks listed under Section 3.1 are straightforward variations of well known graph traversal techniques. They seem quite modest in computational cost, considering the relatively unconstrained nature of the data structure described above.

Compilation can be performed by instantiating frames for each of the entities described by a model. The precise way in which this is done varies depending on the implementation. However, as noted in the previous section, the use of globals indexed by strings in MUMPS provides descriptive power equivalent to structures and property lists in LISP. In either implementation it would appear that the cost is linear in both space and time[15].

Simulation can be performed by breadth-first propagation of perturbations among parameters as follows:

1. *Initialize:* Set all parameter values to normal and the status of all processes to active.

2. Obtain, from the user, the perturbations to parameters and impairments of processes. Mark these accordingly and enter the perturbed parameters into a FIFO *parameter-queue*.

3. *Propagate:* If there are no parameters in the *parameter-queue* then stop the simulation. Else, for each parameter in the *parameter-queue* add all active processes influenced, at the lowest taxonomic level, to a FIFO *process-queue*. If an impaired process is encountered then inform the user and stop the simulation.

4. Flush the *parameter-queue*.

5. For each processes in the *processes-queue*, if the processes has non-null quantitative-rules and its influencing-parameters all have non-null numeric values, then *proceed numerically*. Else *proceed qualitatively*.

6. Flush the *process-queue*. Go to *propagate*.

---

[15] In the following discussion all estimates of computational cost are with respect to the number of entities described by a model, hence the number of nodes in the graph representing it.

7. To *proceed numerically:* Evaluate each quantitative rule and assign the resulting value to the parameter-influenced. If the value is outside the physiologic range then inform the user and stop the simulation. Else add the parameter-influenced to the *parameter-queue.* Return.

8. To *proceed qualitatively:* Evaluate each qualitative rule (by applying the semantics of the specified qualitative operator) and assign the resulting value to the parameter influenced. If this value is not <unknown> then add the parameter-influenced to the *parameter-queue.* Return.

This breadth-first propagation of perturbations might be exponential in the worst case, but, at any given taxonomic level, there seems to be a very limited branching factor (often 1), and hence only a handful of paths to be followed. Perturbations are propagated as numeric values so long as they, and quantitative-rules to relate them, are available. When these are not available, propagation is continued on a qualitative basis. A trace is kept of the simulation, and questions of *why* something happened are answered by indicating the influencing-process, and its influencing-parameters, which caused a parameter to change value, along with the corresponding quantitative rule where appropriate. For example: "In step n, alveolar pCO_2 (the parameter-influenced) was increased by increased $FICO_2$ (the influencing-parameter) through lower airways gas mixing (the influencing-process)."

Regarding explanation, the description of parameters, processes, and states is computationally straightforward and can be accomplished by filling the following sorts of templates:

1. What is <parameter>?

   <parameter> is a physiologic parameter, the <description> measured in <units>. Its normal values are <lower limit> to <upper limit>, and its physiologic range is <lower limit> to <upper limit>. It is <clinical-measurability> to measure in most clinical settings. Increased <parameter> is called <increased state>. Normal <parameter> is called <normal state>. Decreased parameter is called <decreased state>. For more information see <references>.

2. What is <process>?

   <process> is a <descriptive-level-of-abstraction> process, the <description>. It is a mechanism of <is-a-mechanism-of>, and serves to <teleology>. It mediates the influence of <influencing-parameters> on <parameters-influenced>. Increasing <parameter> [increases, decreases] <parameter>, and vice-versa. The

mechanisms of ⟨process⟩ are ⟨mechanisms⟩. For more information see ⟨references⟩.

3. What is ⟨state⟩?

⟨state⟩ is a ⟨descriptive-level-of-abstraction⟩ state characterized by [[increased, normal, decreased] ⟨parameter⟩, impaired ⟨process⟩]. For more information see ⟨references⟩.

Explanation of static relationships[16] requires an algorithm to find a path formed by influence links between two nodes in the directed graph representing a model. This requires $O(n^2)$ time using the following marking algorithm:

1. *Initialize:* Mark the two nodes between which a path is to be sought, each with its own name. Push these two nodes onto stack *A*. Create an empty stack, *B*.

2. *Iterate:* If stack A is empty then stop, there is no path between the two nodes.

3. For each node in stack A (influencing node), examine each node to which it has an incoming or outgoing influence link (influenced node).

4.

   a. If the influenced node is not marked then mark it with a list of names created by concatenating its name to the mark of the influencing node. Push the influenced node onto stack B.

   b. If the influenced node is marked and if the first name in the mark lists of the influenced and influencing nodes are not equal, then a path has been found. The path is described by the concatenation of the mark list of the influencing node and the reversal of the mark list of the influenced node.

5. Interchange stacks A and B. Flush stack B. Go to *Iterate*.

This procedure may be carried out one taxonomic level at a time in order to provide increasingly detailed or increasingly abstract explanations. The limited integrity checking referred to in Section 3.1 also requires verification that a path exists, at a given taxonomic level, between two nodes. It too can be performed using this same marking algorithm.

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[16] Answering the questions listed in Section 3.1 (What is directly influenced by, etc.).

4.5. Evaluation Techniques

The preliminary evaluation of KBPMS was carried out by the author in collaboration with the director of the "Matter and Energy" segment of the New Pathway curriculum. The performance of the final version of KBPMS and the contents of the respiratory model described in Section 4.2 were submitted for review, and subsequently approved by him. The author prepared a syllabus of educational objectives, a homework exercise, and an evaluation quiz in respiratory physiology which were also reviewed and modified by the curriculum director. These are shown in Appendix IV. The quiz contained 8 questions which were equally divided into two different types. Type I questions (1, 3, 5, and 7) were worded so as to be very similar to the questions which might be asked of the KBPMS program. Type II questions (2, 4, 6, and 8) were worded to avoid any such similarity.

In January 1986, during the regularly scheduled pulmonary physiology segment of the New Pathway curriculum, the students were given the homework assignment. A randomly selected half of the group was also given a diskette containing the KBPMS program and the respiratory model along with instructions on how to install and use these on their HP-150 microcomputers. These students were asked to use KBPMS as an educational resource, along with their textbooks, class notes, and laboratory material, in completing the homework assignment. The students were not requested to hand in the homework and it was never reviewed or graded. All the students were fully aware of the experiment in progress and their co-operation was solicited on an entirely voluntary basis. They were carefully informed that the results of the experiment would in no way influence their own evaluation in the physiology course.

Ten days after distributing the homework assignments to all students, and the diskettes to half of them, everyone was given the evaluation quiz and asked to do it at home without referring to any educational resources (closed-book, closed-computer). Their collaboration was, once again, to be wholly voluntary. Following the quiz, the remaining half of the class was given KBPMS diskettes. On several different occasions, all the students were encouraged to submit their evaluation of the program's pedagogic value.

One month after the quizzes were handed out to the 24 students, 8 of them had been completed and returned. Of these, 5 came from the experimental group (those students with access to KBPMS while doing the homework) and 3 came from the control group (those

students who were given KBPMS only after the homework and quiz). In the same one month period, 9 students submitted their evaluations of KBPMS.

5. Results

This chapter presents the results of the KBPMS project. It describes the prototype and final implementations of the intelligent physiologic modeling system as well as the outcome of the evaluation experiment.

5.1. The Prototype Implementation

The prototype of the intelligent physiologic modeling system was constructed in MACLISP [44] and runs on a DEC-1080 mainframe in the MIT Laboratory for Computer Science. The prototype implements all of the specified design features except for impaired physiologic processes. It has been fully debugged and tested using the model of respiratory physiology discussed in Section 4.2. When running that model, the prototype provides under two second turnaround time for both simulation and explanation. Timing studies indicate that the majority of computational overhead is incurred by string manipulation and output formatting, rather than by the reasoning components of the program[17]. Approximately one month of full time programming effort was required to construct and debug the prototype.

5.2. The Final Implementation

The final version of KBPMS was constructed in DataTree PC/MUMPS/2.1 (DT-MUMPS) [45] and runs on the New Pathway's HP-150 microcomputers. It implements all of the specified design features (including impaired processes), has been fully debugged, tested, and was used by the New Pathway students with the respiratory model shown in Appendix I. When running that model, KBPMS provides 15 to 30 second turnaround time for simulation and 3 to 5 second turnaround for explanation. Timing studies indicate that much of this overhead is attributable to graphics-related computation and, like the prototype, reasoning components of the program incur relatively modest computational cost[18]. Approximately three months of full time programming effort were required to complete the final version. Appendix II lists the instructions for using the final version of KBPMS and Appendix III shows an interactive session which illustrates both its reasoning

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[17] It should be noted that MACLISP has extremely primitive string manipulation capabilities.

[18] DT-MUMPS running on the HP-150 has very slow mathematical and trigonometric functions. These are principally responsible for the long graphics computation times.

and graphics capabilities[19].

5.3. Evaluation Results

As mentioned previously, all 24 New Pathway students were asked to do the evaluation quiz on a voluntary basis and 8 of them returned the completed quiz to their instructor. Of the 8 quizzes, 5 came from the experimental group (KBPMS access) and 3 came from the control group (no program access). Table 5-1 summarizes the quiz scores for the two groups. The raw data is presented in Appendix V. Type I questions are those which were worded to resemble KBPMS, Type II questions are those which were worded to be dissimilar to it.

The results shown in Table 5-1 indicate that the experimental group scored very slightly higher than the control group overall. This relationship holds for Type II questions considered alone, but is reversed for Type I questions considered alone. These results are not statistically significant because of the very small differences and sample size. Clearly, no inferences as to the pedagogic value of KBPMS may be drawn from this data.

As indicated previously, there was a wide variety of reactions to KBPMS among the 9 students who submitted their evaluations of it. Some felt that it was an unnecessarily elaborate way to teach very simple concepts. Others stated that the material covered was far too advanced, or might be suitable only as a final review aid rather than a primary learning resource. There were several comments that occupied an intermediate position in this spectrum of opinions. Appendix VI shows some of the student comments which were representative of each extreme as well as the middle ground. There was no uniform consensus among the students' opinions.

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[19] Unfortunately, several important features such as the use of the touch sensitive screen for interactive graphics are not amenable to presentation in this printed medium.

(all scores are %)

Question	Type	Experimental (n=5) mean	Experimental (n=5) s.d.	Control (n=3) mean	Control (n=3) s.d.	Both (n=8) mean	Both (n=8) s.d.
1	I	76	24	93	12	83	21
2	II	66	42	43	40	58	40
3	I	75	18	67	29	72	21
4	II	90	10	97	6	93	9
5	I	80	27	77	25	79	25
6	II	74	37	100	0	84	31
7	I	90	7	93	6	91	6
8	II	72	23	40	35	60	30
All Type I Questions		80	20	83	21	81	20
All Type II Questions		76	30	70	37	73	32
All Questions		78	25	76	30	77	27

Table 5-1: Summary of Quiz Scores

6. Discussion

This chapter begins by describing some of the strengths and weaknesses of the methods used in constructing KBPMS and their effects on the program. This is followed by a discussion of the outcome of the evaluation experiment and a consideration of possible directions for future work suggested by this project. The chapter concludes by discussing some of the non-technical implications of this work.

6.1. The Strengths of KBPMS

The principal advantage of the representational system employed by KBPMS is that the taxonomy based on processes' levels of abstraction allows for explicit control of the depth at which reasoning for simulation and explanation takes place. This potential has been fruitfully exploited in the explanation section of KBPMS. This is illustrated by Figure 6-1 which shows a question being answered at several different levels of detail[20]. However, the potential power of the knowledge representation has not been fully exploited in this regard by the simulation section. KBPMS, like some of its predecessors (eg. NEPHROS [40]), performs simulation by the propagation of perturbations at only the most detailed taxonomic level.

The major advantage of the simulation algorithm used in KBPMS is its ability to combine numeric and qualitative values. As shown by the simulation in Figure 6-2, numeric values are used whenever they are available and appropriate, with qualitative values being used otherwise. This approach is consonant with both clinical and teaching practice when only incomplete numeric data is available, as in most real-world situations.

Another strength of KBPMS is its representation of physiologic entities as having meaningful internal structure. The examination of this internal structure is the basis of the program's explanation capabilities. The need for sound explanations based on physiologic principles has been one of the driving forces in recent MAI research, as discussed in Section 2.2. Certainly the importance of explanations cannot be overlooked in a teaching setting.

The simulation and explanation algorithms described in Section 4.4 have been quite

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[20] An interesting related point is whether the strategy of generating such explanations in a top-down manner (most abstract explanation first), as done by KBPMS, is indeed pedagogically preferable to the inverse strategy.

Figure 6-1: A Question Being Answered at Several Different Levels of Detail

Q: What directly influences CO2 excretion rate (VCO2) ?

A: This question may be answered at four different levels of detail. At the most abstract level, CO2 excretion rate is directly influenced by metabolic CO2 production rate through CO2 elimination. At a more detailed level, CO2 excretion rate is directly influenced by arterial pCO2 through respiratory control of pCO2. At an even more detailed level, CO2 excretion rate is directly influenced by respiratory rate and tidal volume through pulmonary gas exchange. At an even more detailed level, CO2 excretion rate is directly influenced by alveolar pCO2 and alveolocapillary CO2 diffusion rate through alveolocapillary diffusion.

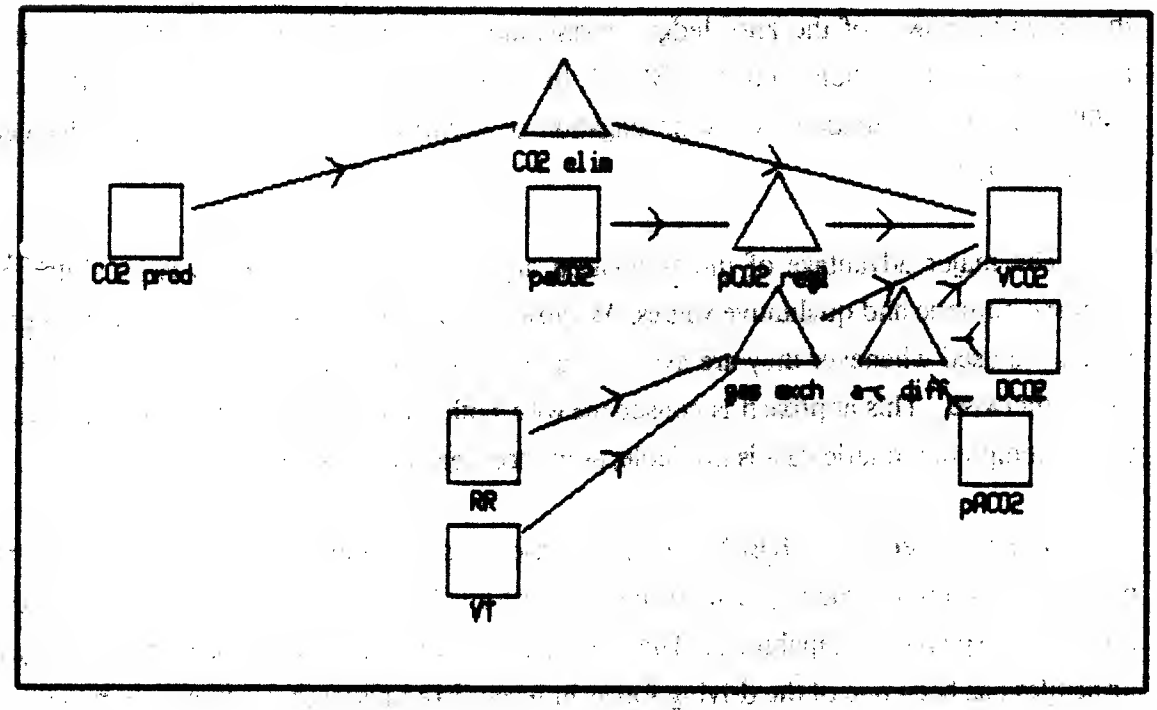
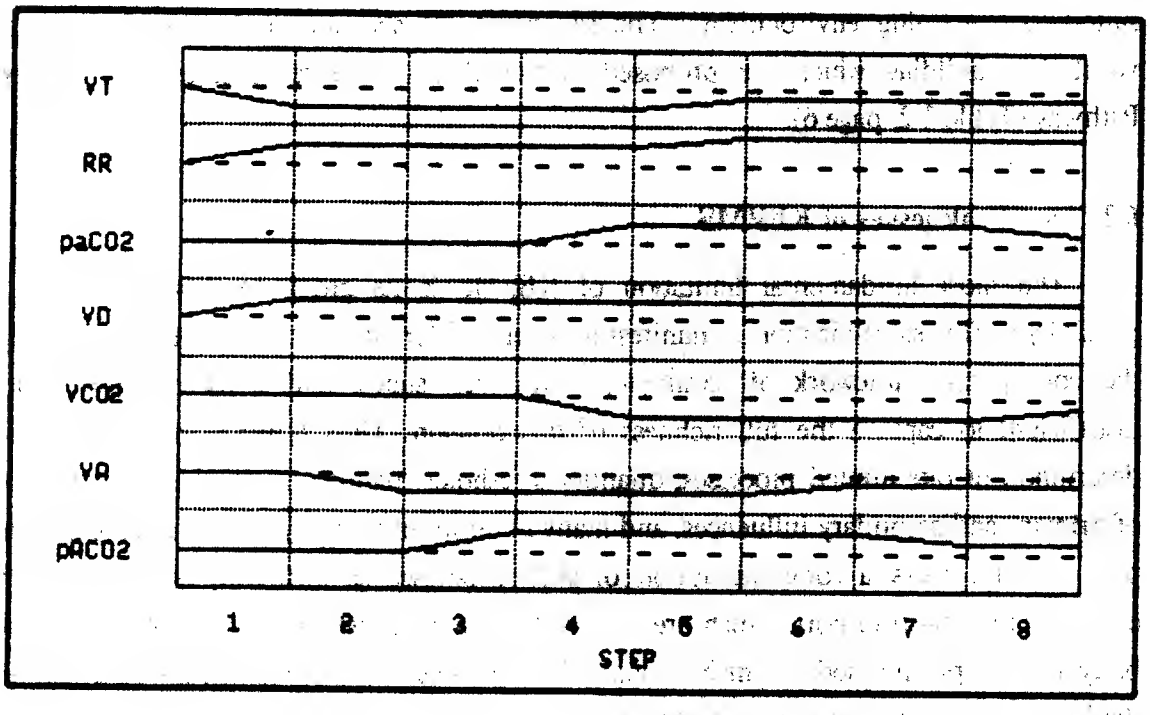


Figure 6-2: A Simulation Showing Mixed Numeric and Qualitative Capabilities

In step 1, dead space=250 ml (increased), respiratory rate=20 breaths per min (increased) [ie. hyperpnea] and tidal volume=350 ml (decreased) as specified by the user.
In step 2, bulk gas flow mediates the following influence: VA=RR*(VT-VD). Since respiratory rate=20 breaths per min (increased), tidal volume=350 ml (decreased) and dead space=250 ml (increased), therefore alveolar ventilation=2000 ml per min (decreased) [ie. alveolar hypoventilation].
In step 3, alveolar pCO2 is increased by decreased alveolar ventilation (2000 ml per min) through lower airway gas mixing.
In step 4, CO2 excretion rate is decreased and arterial pCO2 is increased [ie. hypercapnea] by increased alveolar pCO2 through alveolocapillary diffusion.
In step 5, respiratory rate is further increased [ie. augmented hyperpnea] and tidal volume is increased toward normal by increased arterial pCO2 through the medullary chemoreflex.
In step 6, alveolar ventilation is increased toward normal [ie. diminished alveolar hypoventilation] by increased respiratory rate and increased tidal volume through bulk gas flow.
In step 7, alveolar pCO2 is decreased toward normal by increased alveolar ventilation through lower airway gas mixing.
In step 8, CO2 excretion rate is increased toward normal and arterial pCO2 is decreased toward normal [ie. diminished hypercapnea] by decreased alveolar pCO2 through alveolocapillary diffusion.



efficient in practice. The reasoning features of both the preliminary and final versions of KBPMS have incurred only minor computational costs relative to far more mundane features of the programs, such as string manipulation and graphics respectively. This would tend to confirm the intuition that KBPMS is suitable for modeling several overlapping aspects of human physiology, each of which might be on the same order of magnitude as the present respiratory model.

An advantage which is closely related to efficiency is the simple overall structure of the modeling system. This has permitted its implementation using standard programming technology (ie. an imperative, procedural, programming language, without specific list processing capabilities) following its initial development and refinement in a LISP environment. This is an important consideration in view of the resource and compatibility constraints imposed by the microcomputer setting in which the program was to be ultimately used.

Other advantages of the intelligent modeling system include its ability to accommodate synonyms and its friendly user interface which incorporates interactive graphics. Together, these features greatly enhance the flexibility which seems particularly suited to a learning environment. The synonyms are also appropriate to the task of vocabulary building which was proposed as one of the software modules for the New Pathway (Table 1-2, page 6).

6.2. The Weaknesses of KBPMS

The most fundamental limitation of KBPMS is its gross simplification of real physiology. This simplification is manifest in several different ways. Most important is that the conceptual framework of parameters, processes, states, and steps is substantially insufficient to capture the full richness of this domain. Thus KBPMS has no explicit description of rate limited processes, quantity thresholds, parallel mechanisms, interaction of primary and secondary influences, and many other important physiologic concepts. The program also lacks a coherent notion of such processes as diffusion, bulk flow, and electromotive ion transport, which are a more fundamental aspect of physiology than the body-system-specific models which it manipulates. Processes are unrealistically restricted to the binary status of active or impaired, and cannot represent the typically partial or altered functionality associated with most pathological conditions.

Simplification is also a liability in both the quantitative and qualitative simulation methods employed by KBPMS. Numerical operations are permitted on only the static values of parameters, and the program therefore lacks the full descriptive power of a differential equation model. Qualitative reasoning is restricted to the application of two operators relating eight possible values. Furthermore, the definitions of these values blur the distinctions between the magnitude, direction of change, rate of change, and the past history of a parameter. Temporal relationships beyond simple ordering are entirely ignored.

States are also greatly simplified and allow for the description of only a single perturbed parameter or a single impaired process. In reality, a pathological state such as adult respiratory distress syndrome is associated with a large number of such perturbations and impairments.

The limitation of carrying out simulation at only the most detailed taxonomic level was referred to in the previous section. A consequence of this is the program's inappropriate response to the impairment of processes which are at a more abstract level. For example, if the process of CO_2 elimination shown in Figure 4-4 (page 23) was impaired but the other processes shown in the Figure were active, simulation would proceed unaffected by the impairment. A possible remedy would be to propagate all impairments through to the leaves of their taxonomic trees. However, in this example, as in most situations, this would merely cause simulation to stop altogether as all pertinent processes would be impaired.

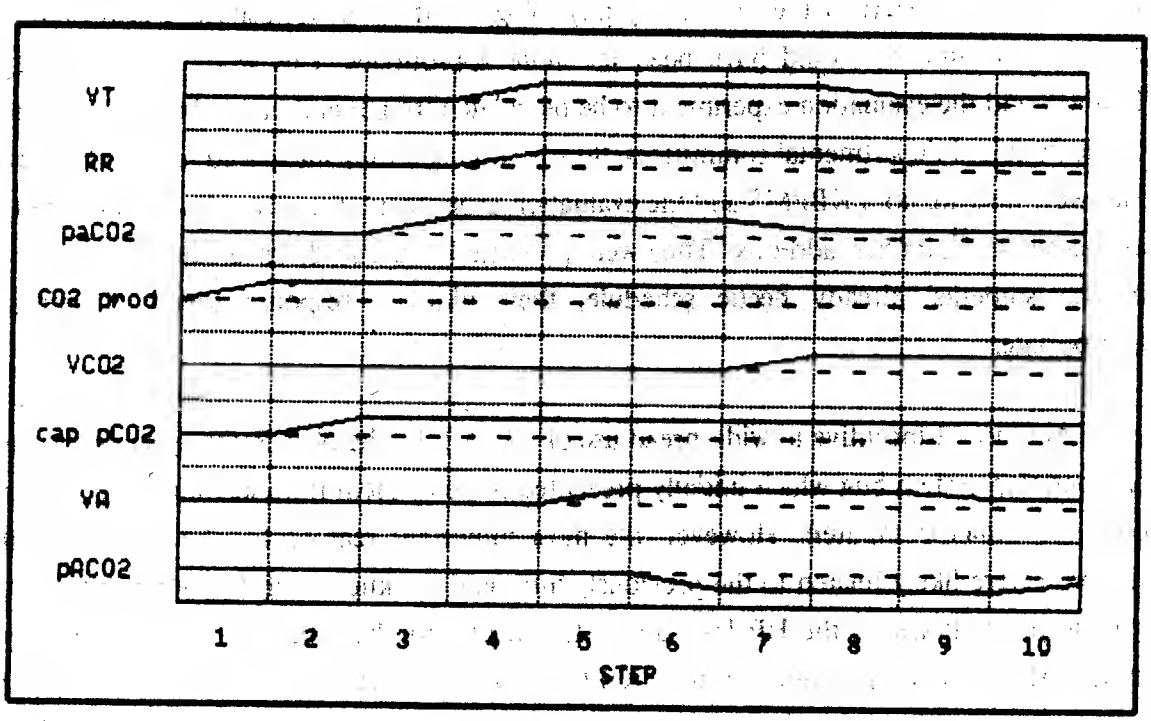
Another weakness of the process taxonomy is that it is not really strict. A low level process such as diffusion is a common mechanism of several more abstract ones. The desire to maintain a strict taxonomy necessitates the somewhat contrived definition of tissue capillary CO_2 diffusion, alveolocapillary diffusion, and lower airways gas mixing as distinct entities (processes). Clearly, each of these is fundamentally the same physical phenomenon, but that knowledge is not encoded in either KBPMS or the respiratory model.

A specific weakness of the present respiratory model, which is not an inherent limitation of KBPMS, is the lack of separate descriptions of the arterial, capillary, and venous portions of the systemic and pulmonary hematogenous circulations. The current respiratory model implicitly assumes that CO_2 is exchanged between the alveoli and the

systemic arteries. This simplifying assumption leads to specific inaccuracies in simulation. For example, if one starts a simulation by increasing metabolic CO_2 production rate, as in Figure 6-3, then the program shows that alveolar pCO_2 ($pACO_2$) is decreased in step 6. Each inference made from step 1 to step 6 is correct but the result is not. The model is incomplete and neglects to consider the increased pCO_2 in systemic veins, hence in pulmonary arteries and capilleries, which would diffuse into the alveoli and increase $pACO_2$.

Figure 6-3: An Incorrect Simulation

In step 1, metabolic CO2 production rate is increased as specified by the user.
In step 2, capillary pCO2 is increased by increased metabolic CO2 production rate through tissue-capillary CO2 diffusion.
In step 3, arterial pCO2 is increased [ie. hypercapnea] by increased capillary pCO2 through circulatory flow.
In step 4, respiratory rate is increased [ie. hyperpnea] and tidal volume is increased by increased arterial pCO2 through the medullary chemoreflex.
In step 5, alveolar ventilation is increased [ie. alveolar hyperventilation] by increased respiratory rate and increased tidal volume through bulk gas flow.
In step 6, alveolar pCO2 is decreased by increased alveolar ventilation through lower airway gas mixing.
In step 7, CO2 excretion rate is increased and arterial pCO2 is decreased toward normal [ie. diminished hypercapnea] by decreased alveolar pCO2 through alveolocapillary diffusion.
In step 8, respiratory rate is decreased toward normal [ie. diminished hyperpnea] and tidal volume is decreased toward normal by decreased arterial pCO2 through the medullary chemoreflex.
In step 9, alveolar ventilation is decreased toward normal [ie. diminished alveolar hyperventilation] by decreased respiratory rate and decreased tidal volume through bulk gas flow.
In step 10, alveolar pCO2 is increased toward normal by decreased alveolar ventilation through lower airway gas mixing.



6.3. The Evaluation Experiment

Many of the above strengths and weaknesses of KBPMS were suspected at the start of this project. Less well understood, and therefore of greater interest, were their potential ramifications for the program's utility as a learning resource. Unfortunately, even relative to the modest objectives outlined in Section 3.3, the evaluation experiment must be considered unsuccessful. The meager data which were collected cannot support any substantive conclusions regarding the program's pedagogic value. The most obvious reason for this lack of success was the small number of New Pathway students who complied with the request for voluntary participation in the homework exercise and quiz. This somewhat uncharacteristic lack of enthusiasm was, in turn, the consequence of several distinct causes. One cause was a severe crowding of the students' schedules just when this experiment was underway. Even given that medical students are chronically overburdened, they were much busier than usual at this time.

A second reason for the students' unenthusiastic response was the lack of advance notice given to all concerned regarding KBPMS and the evaluation experiment. The program and evaluation instruments were first submitted to the curriculum director six weeks before the start of the respiratory physiology course. In retrospect, a lead-time in excess of six months would have been far more appropriate. This would have allowed KBPMS and the evaluation experiment to be percolate through and be modified by various curriculum and departmental committees, thus becoming a fully integrated part of the New Pathway. Instead, both KBPMS and the evaluation experiment were regarded by the faculty as last minute[21], *ad hoc*, add-ons. They were presented to the students as such, and in view of the students' already hectic schedule, their ultimate response seems altogether appropriate.

Another disincentive to widespread use of KBPMS by the New Pathway students was an operating system bug which initially prevented some of them from loading the program onto their microcomputers. However, the most important reasons for the poor student response were likely related to the previously indicated weaknesses of KBPMS itself, to its slow response times on the HP-150, and to the overly simplistic nature of the respiratory model. Because of these and because KBPMS had not been fully integrated into their

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[21] In fact, the program was distributed to the students just as they completed their study of pulmonary physiology and moved on to the renal segment of the course.

curriculum, many students did not regard it as sufficiently relevant to their immediate learning needs to merit even the modest investment of time and effort (perhaps half an hour) required to become well acquainted with its use. Furthermore, a detailed laboratory-type exercise which might have "led the students through" the program's capabilities was not provided[22]. Overall, insufficient attention was paid to the pedagogic and student interface issues relating to KBPMS. The failure of this evaluation experiment reinforces the importance of these issues and suggests that they merit increased consideration in future work of this type.

In addition to the small student response, there were a number of serious limitations in the design of the evaluation experiment which might also have diminished the validity of its results. Even if all 24 students had returned the quiz, this would still have been a small sample size, and an unrealistically large difference in scores would have been required for statistical significance[23]. In addition, with such small numbers, the random division of students might not have resulted in an equitable distribution of talent among the two groups. Since both the homework and quiz were carried out under uncontrolled conditions, it is difficult to know how many students really had access to the program, how many students really completed the quiz closed-book, and so forth.

Yet another limitation of this beleaguered evaluation attempt lay in the nature of the outcome being measured. The goals of the New Pathway stress the acquisition of attitudes, skills, and knowledge which might equip physicians for a lifelong learning process. The role of information technology in the New Pathway is to provide resources to facilitate such lifelong learning and to initiate and encourage students in their use. Attempting to evaluate such an effort by measuring the students' knowledge of respiratory physiology ten days after using a simulation program seems irrelevant or even contradictory to the spirit of the entire enterprise. An alternative outlook on evaluation might have been be to consider the modeling system as a small part of a large educational experiment whose global results will not be known for many years or even decades. If the New Pathway eventually succeeds, and if student and faculty response to intelligent physiologic modeling encourage its ongoing

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[22]The homework assignment shown in Appendix IV was much too general for this purpose.

[23]Assuming standard deviations of 20%, a difference of 11% in mean scores would have been required for $p<0.1$, and a difference of 20% would have been required for $p<0.01$, using a one tailed t-test.

use and development, then such modeling will have been demonstrated to be a useful educational aid, and vice-versa. However, this latter outlook does not suggest an effective means of assessing preliminary progress toward the ultimate education goal. Evidently, the meaningful evaluation of this type of project remains a very substantial challenge.

6.4. Directions for Future Work

Viewed from a somewhat more optimistic perspective, the outcome of the evaluation experiment supports the assertion that KBPMS is at least potentially exploitable as a learning resource and, along with the preceding discussion of the program's strengths and weaknesses, suggests the following directions for future work:

- Enhancement of the respiratory model to include separate descriptions of the arterial, venous, and capillary parts of the systemic and pulmonary circulations. It also seems worthwhile to add a description of pO_2 regulation and some pertinent aspects of acid-base metabolism.

- Repetition of a modified evaluation experiment with a longer time span, better student interface materials, and closer collaboration with multiple members of the New Pathway faculty. Evaluation of KBPMS in a different educational setting would also be of interest.

- Development of an authoring module to facilitate the construction of models of other aspects of physiology and the construction of several such models. Cardiovascular, renal, and endocrine physiology all seem well suited for this purpose.

- Enhancement of KBPMS to incorporate more powerful representation and reasoning capabilities. The use of a differential equation model for numerical simulation and a QSIM-like [39] algorithm for qualitative simulation might be good first steps in this direction.

- There is, of course, a potentially enormous amount of work which might be done to advance the frontiers of our ability to understand, describe, model, and simulate physical events in accordance with the causality which we traditionally ascribe to them. Ultimately, this might lead to the development of more powerful techniques upon which one might build a more intelligent physiologic modeling system.

6.5. Non-Technical Implications of this Project

This project has attempted to apply computational techniques and resources to the goal of advancing medical education within the framework of the Harvard New Pathway. Although this work has been both preliminary and limited in scope, it seems worthwhile to consider some potential implications of the course which we have begun to chart. The philosophy and objectives of the New Pathway were described in Chapter 1. Might KBPMS (or its successors) impinge on this philosophy or these objectives beyond the immediate technical domain of the work itself? It would appear that intelligent physiologic modeling has several such broader implications and that at least the following two are directly pertinent to this thesis.

One non-technical implication of this project relates to the New Pathway's objective of teaching fundamental attitudes, skills, and knowledge. By advocating KBPMS as a medical education resource we are attempting to impart not only knowledge of physiologic mechanisms, but also the attitude that these mechanisms are a fundamentally important aspect of medicine. This attitude is certainly concordant with the reductionist traditions of modern biomedical science[24] but, as recognized by the developers of the New Pathway [8] and by the authors of the GPEP Report [7], learning such mechanisms is but one aspect of general medical education. For example, effective prevention, intervention, and rehabilitation for respiratory disease depends not only on the sort of mechanistic understanding of pulmonary physiology which the present KBPMS model might foster, but also on insight into the epidemiologic, sociologic, and behavioural aspects of such things as cigarette smoking, occupational exposure to particulate toxins, and airborne environmental pollutants. Of course the teaching of respiratory physiology, with or without the aid of KBPMS, in no way precludes consideration of epidemiologic, sociologic, or behavioural aspects of medicine; and these subjects have each been given a place in the New Pathway as well as in many traditional medical curricula. None-the-less, it seems prudent to recognize that this project has emphasized and reinforced but a single narrow aspect of medical education and has entirely ignored many other, equally important, aspects of this diverse process.

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[24] In fact, the current primacy of the mechanistic, reductionist, and rationalist viewpoint in medicine has been challenged from several different quarters [46, 47, 48, 49, 50, 51, 52], but a detailed consideration of this debate is beyond the scope of the present discussion.

Another non-technical implication of this work concerns the appropriateness of computational metaphors for physiologic (living) entities. The parameters, processes, states, and steps described in Section 4.1 are a simple example of such a metaphor, and one whose limitations are readily apparent. The schematic of HUMAN [23] shown in Figure 2-1 (page 11) represents a more elaborate example of such a metaphor. How much further might metaphors of this sort be extended? Guyton's Textbook of Medical Physiology [42], one of the references upon which the respiratory model used in this project was based, suggests that computational metaphors of human life are applicable without limit. In this current international standard medical text, Guyton introduces his readers to their topic as follows:

> "**Human Physiology.** In human physiology we attempt to explain the chemical reactions that occur in the cells, the transmission of nerve impulses from one part of the body to another, contraction of the muscles, reproduction, and even the minute details of transformation of light energy into chemical energy to excite the eyes, thus allowing us to see the world. The very fact that we are alive is almost beyond our control, for hunger makes us seek food and fear makes us seek refuge. Sensations of cold make us provide warmth, and other forces cause us to seek fellowship and reproduce. Thus the human being is actually an automaton, and the fact that we are sensing, feeling, and knowledgeable beings is part of this automatic sequence of life..." (p. 2)

Thus the human being is actually an automaton?! Not only is this statement preposterous, but the very need to refute it supports Weizenbaum's concept of the prevalent "madness of our times." [53] (p.227) Because of its application of artificial intelligence techniques to human physiologic simulation, the work of this project might be construed to somehow support such absurd notions. It is therefore important to both recognize and clearly state that it does not.

The above are significant implications which extend well beyond the technical aspects of this project and it would be reckless to lose sight of these broader issues as we become engrossed with the details of our task. As responsible professionals, it behooves us to consider not only how, but also why and at what cost we might proceed with our work.

7. Summary and Conclusions

The project described in this thesis has consisted of the design, implementation, and preliminary evaluation of an intelligent physiologic modeling system for use as a medical education resource. This knowledge based physiologic modeling system (KBPMS) has been developed within the context of the New Pathway, an experimental curriculum at Harvard Medical School. The central goal of the New Pathway is to equip future physicians with a set of attitudes, skills, and knowledge which will prepare them for lifelong professional learning as a prerequisite to competent practice. The role of information technology in the New Pathway is to provide resources to facilitate such lifelong learning and to initiate and encourage students in their use.

The development of KBPMS is based on antecedent work in application of computers to education and in computer based physiologic modeling. In the former area it is most closely related to intelligent computer assisted instruction, tutoring, and coaching systems. In the latter field it draws on aspects of numerical modeling, causal reasoning, and qualitative simulation.

KBPMS is designed to carry out compilation, simulation, explanation, and verification of models describing various aspects of physiology. These models describe physiologic entities and events in a simplistic conceptual context made up of parameters, processes, states, and steps. The entities making up a model are taxonomically structured by their level of descriptive detail. The models are represented in a uniform frame based language and variations of simple graph traversal algorithms are used to carry out the functions of KBPMS.

KBPMS was originally implemented as a prototype in LISP on a large mainframe and subsequently in MUMPS on the New Pathway's HP-150 microcomputers. The final version augments the prototype's capabilities with a friendly user interface incorporating interactive graphics. Both versions have a small model of carbon dioxide homeostasis by the respiratory system.

An evaluation experiment was undertaken to assess the pedagogic utility of KBPMS. A quiz in respiratory physiology was given to all New Pathway students after a randomly selected half of them had completed a homework exercise using the program, while the

other half of the group used standard educational resources. Poor student compliance with this voluntary experiment and numerous methodologic difficulties preclude substantive conclusions based on the data collected. Though anecdotal evidence, along with student and faculty comments, suggest that KBPMS might have potential as a learning resource, that potential remains unevaluated.

The major strengths of KBPMS include the explicit representation of processes' levels of descriptive detail, combined numeric and qualitative simulation capabilities, explanatory powers, and a simple overall structure. The principal weakness of KBPMS is gross and occasionally misleading simplification of real physiology. This project suggests that ample work remains to be done in enhancing both KBPMS and the respiratory model, in constructing models of other areas of physiology, in pursuing a more sophisticated evaluation effort, and in developing new techniques upon which future systems of this type might be built. This work also has significant non-technical implications relating to the nature and purpose of medical education as well as to the appropriateness of computational metaphors applied to living entities.

In conclusion, the results of this project indicate that an intelligent physiologic modeling system can be constructed using the limited resources of a microcomputer. This system may be of potential pedagogic value in a medical education setting, but it needs to be evaluated in both a careful and patient manner. Much work remains to be done in enhancing many aspects of the system and in integrating it into the evolving context of lifelong professional learning.

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Appendix I
The Respiratory Model

The following is the frame based representation of a model of carbon dioxide homeostasis by the respiratory system. This model was prepared using the textbooks of Guyton [42] and West [43]. A formatted copy of the final MUMPS version is shown.

```
ALVEOLAR-HYPERVENTILATION
-------------------------

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physiologic
     NAMES:   alveolar hyperventilation
     PERTURBATION:   increased
                     ALVEOLAR-VENTILATION
     TYPE:   state



ALVEOLAR-HYPOVENTILATION
------------------------

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physiologic
     NAMES:   alveolar hypoventilation
     PERTURBATION:   decreased
                     ALVEOLAR-VENTILATION
     TYPE:   state



ALVEOLAR-PCO2
-------------

     ASSOCIATED-STATES:   NIL
                          NIL
                          NIL
     CLINICAL-MEASURABILITY:   possible but unusual
     DEFAULT-NUMERIC-VALUE:   40
     DEFAULT-QUALITATIVE-VALUE:   normal
     DESCRIPTION:   partial pressure of cabon dioxide in alveolar gas
     GLAB:   pACO2
     ICON:   .96875
             .333333333333
             1
             4
             45
     INFLUENCING-PROCESSES:   PULMONARY-GAS-TRANSPORT
                              LOWER-AIRWAY-GAS-MIXING
     NAMES:   alveolar pCO2
              pACO2
     NORMAL-RANGE:   38
                     42
     PHYSIOLOGIC-RANGE:   15
                          100
     PROCESSES-INFLUENCED:   ALVEOLOCAPILLARY-DIFFUSION
```

```
     REFERENCES:   Guyton pp. 495-496
                   West pp. 15-17
     TYPE:   parameter
     UNITS:   mm Hg


ALVEOLAR-VENTILATION
--------------------

     ASSOCIATED-STATES:   ALVEOLAR-HYPERVENTILATION
                          NIL
                          ALVEOLAR-HYPOVENTILATION
     CLINICAL-MEASURABILITY:   possible but unusual
     DEFAULT-NUMERIC-VALUE:   4200
     DEFAULT-QUALITATIVE-VALUE:   normal
     DESCRIPTION:   rate at which new air enters the alveoli
     GLAB:   VA
     ICON:   .7
             0
             1
             4
             45
     INFLUENCING-PROCESSES:   BULK-GAS-FLOW
     NAMES:   alveolar ventilation
              VA
     NORMAL-RANGE:   3000
                     7000
     PHYSIOLOGIC-RANGE:   1000
                          10000
     PROCESSES-INFLUENCED:   LOWER-AIRWAY-GAS-MIXING
     REFERENCES:   Guyton pp. 484-486
                   West pp. 15-17
     TYPE:   parameter
     UNITS:   ml per min


ALVEOLOCAPILLARY-CO2-DIFFUSION-RATE
-----------------------------------

     ASSOCIATED-STATES:   NIL
                          NIL
                          NIL
     CLINICAL-MEASURABILITY:   possible but unusual
     DEFAULT-NUMERIC-VALUE:   NIL
     DEFAULT-QUALITATIVE-VALUE:   normal
     DESCRIPTION:   rate at which carbon dioxide difuses accross the alveoloca
                    pillary basement membrane
     GLAB:   DCO2
     ICON:   1
             .5
             1
             4
             45
     INFLUENCING-PROCESSES:   NIL
     NAMES:   alveolocapillary CO2 diffusion rate
              DCO2
     NORMAL-RANGE:   NIL
     PHYSIOLOGIC-RANGE:   NIL
```

PROCESSES-INFLUENCED: ALVEOLOCAPILLARY-DIFFUSION

TYPE: parameter
UNITS: ml per min

ALVEOLOCAPILLARY-DIFFUSION

ASSOCIATED-STATES: ARDS
DEFAULT-STATUS: active
DESCRIPTION: passive diffusion of gas between the alveoli and the
capillary blood

DESCRIPTIVE-LEVEL-OF-ABSTRACTION:
GLAB: a-c diff
ICON:

INFLUENCING-PARAMETERS:

IS-A-MECHANISM-OF:
MECHANISMS: NIL
NAMES: alveolocapillary diffusion
a-c diff
PARAMETERS-INFLUENCED: CO2-EXCRETION-RATE
PCO2
QUALITATIVE-RULES:

QUANTITATIVE-RULES: NIL
REFERENCES: Guyton pp. 407-408
West pp. 12-20
TAXONOMIC-LEVEL: 4
TELEOLOGY: NIL
TYPE: process

ARDS

DESCRIPTIVE-LEVEL-OF-ABSTRACTION:
NAMES: ARDS
adult respiratory distress
shock lung
PERTURBATION: Shunted
ALVEOLOCAPILLARY-DIFFUSION
TYPE: state

BULK-GAS-FLOW

ASSOCIATED-STATES: COPD

```
    DEFAULT-STATUS:   active
    DESCRIPTION:   flow of gas volumes through the tracheobrochial tree
    DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physical
    GLAB:   gas flow
    ICON:   .63126
            .166666666667
            1
            3
            90
    INFLUENCING-PARAMETERS:   RESPIRATORY-RATE
                              TIDAL-VOLUME
                              DEAD-SPACE
    IS-A-MECHANISM-OF:   PULMONARY-GAS-TRANSPORT
    MECHANISMS:   NIL
    NAMES:   bulk gas flow
             gas flow
    PARAMETERS-INFLUENCED:   ALVEOLAR-VENTILATION
    QUALITATIVE-RULES:   RESPIRATORY-RATE->M+:ALVEOLAR-VENTILATION
                         TIDAL-VOLUME->M+:ALVEOLAR-VENTILATION
                         DEAD-SPACE->M-:ALVEOLAR-VENTILATION
    QUANTITATIVE-RULES:   'ALVEOLAR-VENTILATION'='RESPIRATORY-RATE'*('TIDAL-V
                          OLUME'-'DEAD-SPACE')
    REFERENCES:   Guyton pp. 484-486
                  West pp. 15-19
    TAXONOMIC-LEVEL:   6
    TELEOLOGY:   NIL
    TYPE:   process


CAPILLARY-PCO2
--------------

    ASSOCIATED-STATES:   NIL
                         NIL
                         NIL
    CLINICAL-MEASURABILITY:   possible but unusual
    DEFAULT-NUMERIC-VALUE:   NIL
    DEFAULT-QUALITATIVE-VALUE:   normal
    DESCRIPTION:   partial pressure of carbon dioxide in the systemic capilla
                   ry  blood
    GLAB:   cap pCO2
    ICON:   .2126
            .5
            1
            4
            45
    INFLUENCING-PROCESSES:   TISSUE-CAPILLARY-CO2-DIFFUSION
    NAMES:   capillary pCO2
             systemic capillary pCO2
             cap pCO2
    NORMAL-RANGE:   NIL
    PHYSIOLOGIC-RANGE:   NIL
    PROCESSES-INFLUENCED:   CIRCULATORY-FLOW
    REFERENCES:   Guyton pp. 506-507
                  West pp. 72-74
    TYPE:   parameter
    UNITS:   mm Hg
```

CHEMOREFLEXES

ASSOCIATED-STATES: NIL
DEFAULT-STATUS: active
DESCRIPTION: built from a hybrid of [illegible]
a group in the [illegible]
DESCRIPTIVE-LEVEL-OF-ABSTRACTION: physiologic
GLAB: chemoref
ICON: .8376
.6
1
3
00
INFLUENCING-PARAMETERS: PCO2
IS-A-MECHANISM-OF: RESPIRATORY-CONTROL-OF-PCO2
MECHANISMS: NIL
NAMES: the medullary chemoreflex
chemoreflex
chemoref
PARAMETERS-INFLUENCED: RESPIRATORY-RATE
TIDAL-VOLUME
QUALITATIVE-RULES: PCO2-XM-[illegible]
QUANTITATIVE-RULES: NIL
REFERENCES: [illegible] transport metabolic substrates and [illegible]
body
West pp. 113-126
TAXONOMIC-LEVEL: 3
TELEOLOGY: detect and mediate a respiratory response to changing levels
of arterial pCO2
TYPE: process

CIRCULATORY-CO2-TRANSPORT

ASSOCIATED-STATES: NIL
DEFAULT-STATUS: active
DESCRIPTION: removal of the carbon dioxide, a waste metabolic byprod
duct, from the body
DESCRIPTIVE-LEVEL-OF-ABSTRACTION: physiologic
GLAB: CO2 trans
ICON: .2126
.666666666667
1
3
00
INFLUENCING-PARAMETERS: METABOLIC-CO2-PRODUCTION-RATE
IS-A-MECHANISM-OF: NONSPECIFICS
MECHANISMS: CIRCULATORY-CO2-TRANSPORT
RESPIRATORY-CO2-[illegible]
NAMES: CO2 elimination
CO2 elim
PARAMETERS-INFLUENCED: CO2-[illegible]
QUALITATIVE-RULES: METABOLIC-CO2-PRODUCTION-[illegible]
QUANTITATIVE-RULES: NIL
REFERENCES: Guyton pp. 612-614
West pp. 72-75
TAXONOMIC-LEVEL: 2
TELEOLOGY: NIL
TYPE: process

```
CIRCULATORY-FLOW
----------------

     ASSOCIATED-STATES:   NIL
     DEFAULT-STATUS:   active
     DESCRIPTION:   bulk flow of blood through the circulatory system
     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physical
     GLAB:   circ flow
     ICON:   .3875
             .6
             1
             3
             90
     INFLUENCING-PARAMETERS:   CAPILLARY-PCO2
     IS-A-MECHANISM-OF:   CIRCULATORY-CO2-TRANSPORT
     MECHANISMS:   NIL
     NAMES:   circulatory flow
              circ flow
     PARAMETERS-INFLUENCED:   PCO2
     QUALITATIVE-RULES:   CAPILLARY-PCO2->M+:PCO2
     QUANTITATIVE-RULES:   NIL
     REFERENCES:   Guyton pp. 504-516
                   West pp. 67-83
     TAXONOMIC-LEVEL:   3
     TELEOLOGY:   transport metabolic substrates and byproducts throughout the
                  body
     TYPE:   process


CO2-ELIMINATION
---------------

     ASSOCIATED-STATES:   NIL
     DEFAULT-STATUS:   active
     DESCRIPTION:   removal of the carbon dioxide, a universal metabolic bypro
                    duct, from the body
     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physiologic
     GLAB:   CO2 elim
     ICON:   .475
             .833333333333
             1
             3
             90
     INFLUENCING-PARAMETERS:   METABOLIC-CO2-PRODUCTION-RATE
     IS-A-MECHANISM-OF:   HOMEOSTASIS
     MECHANISMS:   CIRCULATORY-CO2-TRANSPORT
                   RESPIRATORY-CONTROL-OF-PCO2
     NAMES:   CO2 elimination
              CO2 elim
     PARAMETERS-INFLUENCED:   CO2-EXCRETION-RATE
     QUALITATIVE-RULES:   METABOLIC-CO2-PRODUCTION-RATE->M+:CO2-EXCRETION-RATE
     QUANTITATIVE-RULES:   NIL
     REFERENCES:   NIL
     TAXONOMIC-LEVEL:   1
     TELEOLOGY:   NIL
     TYPE:   process
```

```
CO2-EXCRETION-RATE
------------------

     ASSOCIATED-STATES:    NIL
                           NIL
                           NIL
     CLINICAL-MEASURABILITY:    possible but unusual
     DEFAULT-NUMERIC-VALUE:    NIL
     DEFAULT-QUALITATIVE-VALUE:    normal
     DESCRIPTION:    volume of CO2 exhaled per unit time
     GLAB:    VCO2
     ICON:    1
              .666666666667
              1
              4
              45
     INFLUENCING-PROCESSES:    CO2-ELIMINATION
                               RESPIRATORY-CONTROL-OF-PCO2
                               PULMONARY-GAS-EXCHANGE
                               ALVEOLOCAPILLARY-DIFFUSION
     NAMES:    CO2 excretion rate
               VCO2
     NORMAL-RANGE:    NIL
     PHYSIOLOGIC-RANGE:    NIL
     PROCESSES-INFLUENCED:    NIL
     REFERENCES:    West p. 16
     TYPE:    parameter
     UNITS:    l per min


COPD
----

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:    pathophysiologic
     NAMES:    COPD
               chronic obstructive pulmonary disease
     PERTURBATION:    impaired
                      BULK-GAS-FLOW
     TYPE:    state


DEAD-SPACE
----------

     ASSOCIATED-STATES:    NIL
                           NIL
                           NIL
     CLINICAL-MEASURABILITY:    possible but unusual
     DEFAULT-NUMERIC-VALUE:    150
     DEFAULT-QUALITATIVE-VALUE:    normal
     DESCRIPTION:    volume of tidal gas which does not reach gas-exchanging ar
                     eas of the lung
     GLAB:    VD
     ICON:    .3875
              0
              1
              4
              45
```

```
     INFLUENCING-PROCESSES:   NIL
     NAMES:   dead space
              physiologic dead space
              VD
     NORMAL-RANGE:   100
                     200
     PHYSIOLOGIC-RANGE:   50
                          1000
     PROCESSES-INFLUENCED:   BULK-GAS-FLOW
     REFERENCES:   Guyton pp. 484-486
                   West pp. 15-19
     TYPE:   parameter
     UNITS:   ml


FICO2
-----

     ASSOCIATED-STATES:   NIL
                          NIL
                          NIL
     CLINICAL-MEASURABILITY:   possible but unusual
     DEFAULT-NUMERIC-VALUE:   .04
     DEFAULT-QUALITATIVE-VALUE:   normal
     DESCRIPTION:   fraction of inspired gas volume which is CO2
     GLAB:   FICO2
     ICON:   .96875
             0
             1
             4
             45
     INFLUENCING-PROCESSES:   NIL
     NAMES:   FICO2
              fractionional inspired CO2
     NORMAL-RANGE:   .04
                     .04
     PHYSIOLOGIC-RANGE:   NIL
     PROCESSES-INFLUENCED:   LOWER-AIRWAY-GAS-MIXING
     REFERENCES:   Guyton 493-494
     TYPE:   parameter
     UNITS:   %


HOMEOSTASIS
-----------

     DEFAULT-STATUS:   active
     DESCRIPTION:   regulation of the internal environment by a living organis
                    m
     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physiologic
     GLAB:   homeostasis
     ICON:   .475
             1
             1
             3
             90
     INFLUENCING-PARAMETERS:   NIL
     IS-A-MECHANISM-OF:   survival
```

```
MECHANISMS:   CO2-ELIMINATION
NAMES:   homeostasis
PARAMETERS-INFLUENCED:   NIL
QUALITATIVE-RULES:   NIL
QUANTITATIVE-RULES:   NIL
REFERENCES:   NIL
TAXONOMIC-LEVEL:   0
TELEOLOGY:   maintain those conditions which are compatible with life
TYPE:   summumgenus
```

```
HYPERCAPNEA
-----------

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physiologic
     NAMES:   hypercapnea
              hypercarbia
              hypoventilation
     PERTURBATION:   increased
                     PCO2
     TYPE:   state
```

```
HYPERMETABOLISM
---------------

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   pathophysiologic
     NAMES:   hypermetabolism
     PERTURBATION:   increased
                     METABOLIC-CO2-PRODUCTION-RATE
     TYPE:   state
```

```
HYPERPNEA
---------

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physiologic
     NAMES:   hyperpnea
     PERTURBATION:   increased
                     RESPIRATORY-RATE
     TYPE:   state
```

```
HYPOCAPNEA
----------

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physiologic
     NAMES:   hypocapnea
              hypocarbia
              hyperventilation
     PERTURBATION:   decreased
                     PCO2
     TYPE:   state
```

```
HYPOMETABOLISM
--------------

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   pathophysiologic
     NAMES:   hypometabolism
     PERTURBATION:   decreased
                     METABOLIC-CO2-PRODUCTION-RATE
     TYPE:   state


HYPOPNEA
--------

     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physiologic
     NAMES:   hypopnea
     PERTURBATION:   decreased
                     RESPIRATORY-RATE
     TYPE:   state


LOWER-AIRWAY-GAS-MIXING
-----------------------

     ASSOCIATED-STATES:   NIL
     DEFAULT-STATUS:   active
     DESCRIPTION:   mixing of intrapulmonary gas in the lower tracheobronchial
                    tree
     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physical
     GLAB:   gas mixing
     ICON:   .8625
             .166666666667
             1
             3
             90
     INFLUENCING-PARAMETERS:   ALVEOLAR-VENTILATION
                               FICO2
     IS-A-MECHANISM-OF:   PULMONARY-GAS-TRANSPORT
     MECHANISMS:   NIL
     NAMES:   lower airway gas mixing
              gas mixing
     PARAMETERS-INFLUENCED:   ALVEOLAR-PCO2
     QUALITATIVE-RULES:   ALVEOLAR-VENTILATION->M-:ALVEOLAR-PCO2
                          FICO2->M+:ALVEOLAR-PCO2
     QUANTITATIVE-RULES:   NIL
     REFERENCES:   Guyton pp. 495-496
     TAXONOMIC-LEVEL:   5
     TELEOLOGY:   NIL
     TYPE:   process


METABOLIC-CO2-PRODUCTION-RATE
-----------------------------

     ASSOCIATED-STATES:   NIL
                          NIL
                          NIL
```

```
CLINICAL-MEASURABILITY:   possible but unusual
DEFAULT-NUMERIC-VALUE:   NIL
DEFAULT-QUALITATIVE-VALUE:   normal
DESCRIPTION:   rate at which carbon dioxide is produced by tissue metabol
               ism
GLAB:   CO2 prod
ICON:   0
        .666666666667
        1
        4
        45
INFLUENCING-PROCESSES:   NIL
NAMES:   metabolic CO2 production rate
         CO2 prod
NORMAL-RANGE:   NIL
PHYSIOLOGIC-RANGE:   NIL
PROCESSES-INFLUENCED:   CO2-ELIMINATION
                        CIRCULATORY-CO2-TRANSPORT
                        TISSUE-CAPILLARY-CO2-DIFFUSION
REFERENCES:   Guyton pp. 510-511
TYPE:   parameter
UNITS:   mg per hr per sq cm body surface area
```

PCO2

```
ASSOCIATED-STATES:   HYPERCAPNEA
                     NIL
                     HYPOCAPNEA
CLINICAL-MEASURABILITY:   routine
DEFAULT-NUMERIC-VALUE:   40
DEFAULT-QUALITATIVE-VALUE:   normal
DESCRIPTION:   partial pressure of carbon dioxide in arterial blood
GLAB:   paCO2
ICON:   .475
        .666666666667
        1
        4
        45
INFLUENCING-PROCESSES:   CIRCULATORY-CO2-TRANSPORT,RESPIRATORY-CONTROL-OF
                         -PCO2
                         CIRCULATORY-FLOW,PULMONARY-GAS-EXCHANGE
                         ALVEOLOCAPILLARY-DIFFUSION
NAMES:   arterial pCO2
         pCO2
         paCO2
NORMAL-RANGE:   38
                42
PHYSIOLOGIC-RANGE:   15
                     100
PROCESSES-INFLUENCED:   RESPIRATORY-CONTROL-OF-PCO2
                        CHEMOREFLEXES
REFERENCES:   West p. 1
TYPE:   parameter
UNITS:   mm Hg
```

```
PULMONARY-GAS-EXCHANGE
----------------------

     ASSOCIATED-STATES:    NIL
     DEFAULT-STATUS:    active
     DESCRIPTION:    exchange of gasses between the body and its environment by
                     the lungs
     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:    physiologic
     GLAB:    gas exch
     ICON:    .71875
              .5
              1
              3
              90
     INFLUENCING-PARAMETERS:    RESPIRATORY-RATE
                                TIDAL-VOLUME
     IS-A-MECHANISM-OF:    RESPIRATORY-CONTROL-OF-PCO2
     MECHANISMS:    PULMONARY-GAS-TRANSPORT
                    ALVEOLOCAPILLARY-DIFFUSION
     NAMES:    pulmonary gas exchange
               gas exch
     PARAMETERS-INFLUENCED:    CO2-EXCRETION-RATE
                               PCO2
     QUALITATIVE-RULES:    RESPIRATORY-RATE->M-:PCO2,M+:CO2-EXCRETION-RATE
                           TIDAL-VOLUME->M-:PCO2,M+:CO2-EXCRETION-RATE
     QUANTITATIVE-RULES:    NIL
     REFERENCES:    NIL
     TAXONOMIC-LEVEL:    3
     TELEOLOGY:    NIL
     TYPE:    process


PULMONARY-GAS-TRANSPORT
-----------------------

     ASSOCIATED-STATES:    NIL
     DEFAULT-STATUS:    active
     DESCRIPTION:    transport of tidal gas between the external environment an
                     d the alveoli
     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:    physiologic
     GLAB:    gas trans
     ICON:    .71875
              .333333333333
              1
              3
              90
     INFLUENCING-PARAMETERS:    RESPIRATORY-RATE
                                TIDAL-VOLUME
     IS-A-MECHANISM-OF:    PULMONARY-GAS-EXCHANGE
     MECHANISMS:    BULK-GAS-FLOW
                    LOWER-AIRWAY-GAS-MIXING
     NAMES:    pulmonary gas transport
               pulmonary ventilation
               gas trans
     PARAMETERS-INFLUENCED:    ALVEOLAR-PCO2
     QUALITATIVE-RULES:    RESPIRATORY-RATE->M-:ALVEOLAR-PCO2
                           TIDAL-VOLUME->M-:ALVEOLAR-PCO2
     QUANTITATIVE-RULES:    NIL
     REFERENCES:    Guyton pp. 476-490
                    West pp. 11-20
```

```
     TAXONOMIC-LEVEL:   4
     TELEOLOGY:   NIL
     TYPE:   process


RESPIRATORY-CONTROL-OF-PCO2
---------------------------

     ASSOCIATED-STATES:   NIL
     DEFAULT-STATUS:   active
     DESCRIPTION:   regulation of arterial pCO2 by the respiratory system
     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   physiologic
     GLAB:   pCO2 reg1
     ICON:   .71875
             .666666666667
             1
             3
             90
     INFLUENCING-PARAMETERS:   PCO2
     IS-A-MECHANISM-OF:   CO2-ELIMINATION
     MECHANISMS:   CHEMOREFLEXES
                   PULMONARY-GAS-EXCHANGE
     NAMES:   respiratory control of pCO2
              respiratory pCO2 regulation
              pCO2 reg1
     PARAMETERS-INFLUENCED:   CO2-EXCRETION-RATE
                              PCO2
     QUALITATIVE-RULES:   PCO2->M+:CO2-EXCRETION-RATE,M-:PCO2
     QUANTITATIVE-RULES:   NIL
     REFERENCES:   Guyton pp. 516-527
                   West pp. 113-127
     TAXONOMIC-LEVEL:   2
     TELEOLOGY:   maintain paCO2 at a constant level
     TYPE:   process


RESPIRATORY-RATE
----------------

     ASSOCIATED-STATES:   HYPERPNEA
                          NIL
                          HYPOPNEA
     CLINICAL-MEASURABILITY:   routine
     DEFAULT-NUMERIC-VALUE:   12
     DEFAULT-QUALITATIVE-VALUE:   normal
     DESCRIPTION:   rate of breathing
     GLAB:   RR
     ICON:   .3875
             .333333333333
             1
             4
             45
     INFLUENCING-PROCESSES:   CHEMOREFLEXES
     NAMES:   respiratory rate
              RR
     NORMAL-RANGE:   10
                     15
     PHYSIOLOGIC-RANGE:   5
```

```
                        50
     PROCESSES-INFLUENCED:   PULMONARY-GAS-EXCHANGE
                             PULMONARY-GAS-TRANSPORT
                             BULK-GAS-FLOW
     REFERENCES:   NIL
     TYPE:   parameter
     UNITS:   breaths per min


TIDAL-VOLUME
------------

     ASSOCIATED-STATES:   NIL
                          NIL
                          NIL
     CLINICAL-MEASURABILITY:   possible but unusual
     DEFAULT-NUMERIC-VALUE:   500
     DEFAULT-QUALITATIVE-VALUE:   normal
     DESCRIPTION:   volume of gas expirexd in a siegle breath
     GLAB:   VT
     ICON:   .3875
             .166666666667
             1
             4
             45
     INFLUENCING-PROCESSES:   CHEMOREFLEXES
     NAMES:   tidal volume
              VT
     NORMAL-RANGE:   400
                     600
     PHYSIOLOGIC-RANGE:   50
                          3500
     PROCESSES-INFLUENCED:   PULMONARY-GAS-EXCHANGE
                             PULMONARY-GAS-TRANSPORT
                             BULK-GAS-FLOW
     REFERENCES:   Guyton pp. 480-482
                   West pp. 12-14
     TYPE:   parameter
     UNITS:   ml


TISSUE-CAPILLARY-CO2-DIFFUSION
------------------------------

     ASSOCIATED-STATES:   NIL
     DEFAULT-STATUS:   active
     DESCRIPTION:   passive diffusion of carbon dioxide from metabolizing tiss
                    ues to capillary blood
     DESCRIPTIVE-LEVEL-OF-ABSTRACTION:   chemical
     GLAB:   t-c CO2 diff
     ICON:   .06875
             .5
             1
             3
             90
     INFLUENCING-PARAMETERS:   METABOLIC-CO2-PRODUCTION-RATE
     IS-A-MECHANISM-OF:   CIRCULATORY-CO2-TRANSPORT
     MECHANISMS:   NIL
```

```
NAMES:    tissue-capillary CO2 diffusion
          t-c CO2 diff
PARAMETERS-INFLUENCED:    CAPILLARY-PCO2
QUALITATIVE-RULES:    METABOLIC-CO2-PRODUCTION-RATE->M+:CAPILLARY-PCO2
QUANTITATIVE-RULES:    NIL
REFERENCES:    Guyton pp. 497-500
               West pp. 21-30
TAXONOMIC-LEVEL:    3
TELEOLOGY:    NIL
TYPE:    process
```

APPENDIX II

The following are the instructions

Help:

I. Introduction

exact numeric value is not known (eg. body temperature is increased). Similarly, KBPMS will display numeric values whenever possible, and qualitative values otherwise.

II. General Program Features

KBPMS is designed to run in several different hardware environments and some of the following information may vary depending on the particular implementation. However, most of the general principles should apply.

The video display is divided into four regions in KBPMS. These are: a title line, a framed graphics window, an unframed text window, and a command line (softkeys on the HP-150). The user and the program interact by manipulating the contents of the display windows using the alphanumeric keyboard, the function keys, and pointing mechanisms (a mouse, a touch-sensitive display, the alphanumeric cursor, or some combination of these).

The graphics window may be manipulated by the commands in the Graphics Options menu. The text window may be manipulated (scrolled) by the commands displayed along with the text. For convenience, the four directional arrows (up, down, left, right) and the <Next>, <Prev>, and <Top> (10 o'clock arrow) keys may also be used to augment the function keys and pointing mechanisms. Thus they may be of assistance in scrolling the text window, moving a hot-spot, directing the alphanumeric cursor to point to a symbol, or to request the next step in a simulation. However, they are not required for KBPMS to operate properly and may not be present in some implementations.

When prompted to select a physiologic entity, the user may type its name or a common abbreviation, request a list to choose from (by pressing the space bar), or point to a symbol displayed in the graphics window. The <f8> function key may be used to exit from most segments of the program at any time, whether or not the command line is currently displayed.

KBPMS can be quite slow in performing some of its functions. Please be patient and wait for a response rather than concluding that the program has hung and re-booting your computer.

III. Specific Program Segments and Commands

---> Main Menu <---

- Simulation: Allows the user to carry out an "experiment" by specifying a set of perturbations and observing the physiologic response to them.

- Explanation: Allows the user to ask any of the following questions, where the blanks may be filled by any entity described by the current model:
  . What is _____?
  . What directly influences _____?

. What is directly influenced by _____?
. Does _____ influence _____?
. What are the mechanisms of _____?

- Show Entities: Lists all the entities which are described by the current model and their common abbreviations. The accompanying diagram illustrates the parameters and processes. Note that this command can be particularly slow.

- Logging Off: Terminates logging.

- Logging On: Initiates logging of all textual program output to a file, printer, or other device.

- Graphics Options: Allows the user to magnify, shrink, scroll, and print the currently displayed diagram. This is particularly useful for complex diagrams which may be difficult to read in the ordinary graphics window.

- Help: Displays this information.

- Exit: Leaves KBPMS.

---> Simulation <---

KBPMS first resets all physiologic parameters to their normal values and all physiologic processes to active status. It then prompts the user to specify an entity to perturb. The entity may be selected as described above (II. General Program Features). If the selected entity is a process, then the user is asked to choose one of two possible statuses: active or impaired. If the selected entity is a parameter then the user is asked to choose a qualitative value (normal, increased, or decreased) or a numerical value for the parameter. The user may continue entering additional perturbations, or altering previously entered ones, until he or she decides to start the simulation.

There is a substantial delay while the program prepares the simulation. Afterwards, the user may review the simulation results with the following commands:

- Next Step: Displays the next step of the simulation.

- Prev Step: Displays the previous step of the simulation.

- Why?: Explains why the current simulation step took place.

- Close-up Off: Terminates close-up mode.

- Close-up On: Initiates close-up mode, in which only those entities directly involved in the current simulation step are displayed in the graphics window.

- Graphics Options: Same as in main menu.

- Summary: Displays a graphical and text summary of the entire simulation run and then returns to the main menu.

- Main Menu: Returns to the main menu.

---> Explanation <---

KBPMS asks the user to select one of the available questions and entities to fill in the blank(s) in the selected question. The selection of questions and entities is performed as described above (II. General Program Features). The answer to the question may consist of text and/or diagrams which are displayed after a short delay.

IV. How to get started

The definitions listed above (I. Introduction) are very important. Please review them before you start using KBPMS. A good way to acquaint yourself with both the modeling system and a particular model is to look at the entities described by the model (<Logging On>, log to printer, <Show Entities>; then be prepared to wait, this function is very slow). The summary diagram will seem hopelessly complex and illegible at first. Print a copy (<Graphics Options>, <Print Figure>) and set it aside for future reference. It will help things fall into place as you explore different parts of the model. Go back to the main menu and turn logging off for now. You can turn it back on at any time if you decide that you want to keep a record of your session.

Examine the list of entities. Request explanations of one parameter, one process, and one state that you are already familiar with. Try each of the possible questions to see what sort of answers KBPMS can provide. Now take a look at some entities which are new to you. Don't rely exclusively on the answers provided by the program. Have the suggested references on hand and use them!

Now try the simulation feature. Specify a single perturbation, eg. an increase in one parameter. Examine each step of the simulation and ask "Why?" for each step. Try close-up mode. Carefully examine the summary using the Graphics Options. If the simulation refers to any entities with which you are unfamiliar, go back to explanation mode and inquire about them.

V. Exhortations

Remember that KBPMS is intended as a means of exploration and experimentation. Please try to use it beyond the strict confines of your assignment. If you are unsure of how something works, or whether it works properly, play with it -- see what happens. Whatever "happens", it is hoped that you will learn something about both the program and the particular area of physiology you are studying.

Students, faculty, and staff -- be sure to report your evaluation of KBPMS!!! This feedback is very important from EVERYONE who has occasion to use the program and will be the basis for future modifications. Please report both positive and negative aspects, how long you used the program, and what specific changes you would like to see.

Please send these comments to Robert Kunstaetter via electronic mail using HP-DeskManager. I can also be reached over ARPANET (RKU@MIT-MC) or at the following postal address:

Robert Kunstaetter
Massachusetts Institute of Technology
Laboratory for Computer Science
545 Technology Square, Rm. 373
Cambridge, MA 02139

Have fun!

APPENDIX III

Appendix III
A Sample Interaction

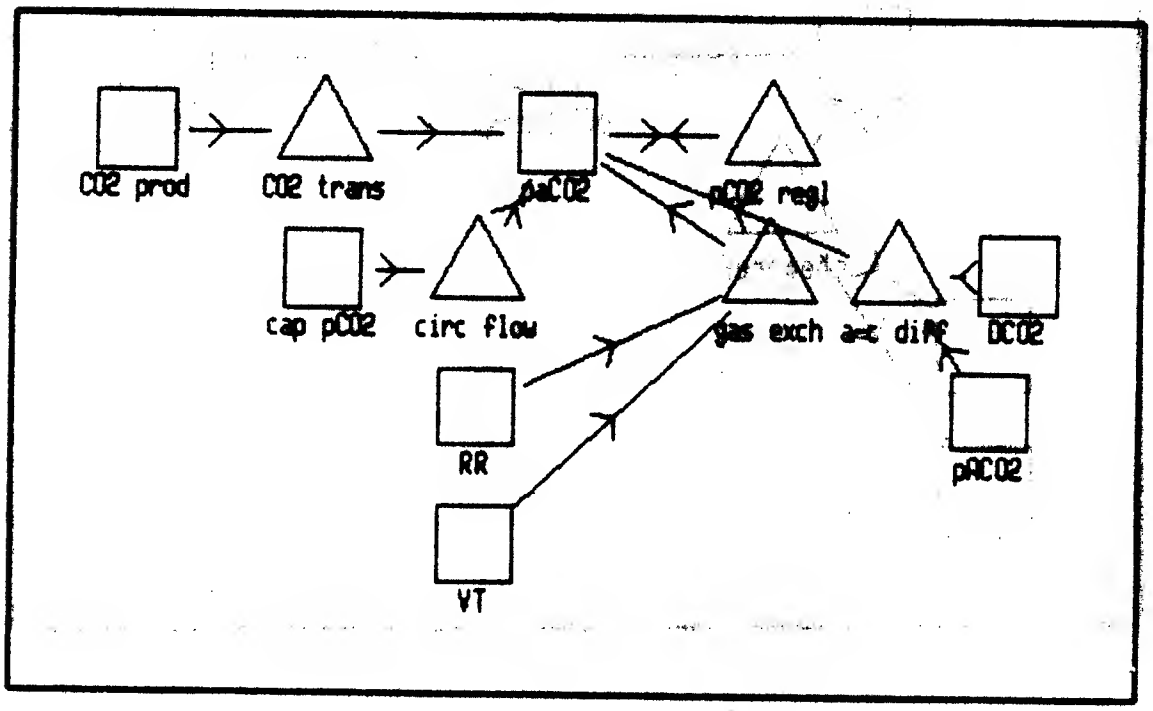
The following is a sample interaction with KBPMS using the respratory model shown in Appendix I. All text and diagrams shown were generated by the program.

```
The following entities are known to this model:

Parameters:
----------
   alveolar pCO2
     --> (a.k.a.: pACO2)
   alveolar ventilation
     --> (a.k.a.: VA)
   alveolocapillary CO2 diffusion rate
     --> (a.k.a.: DCO2)
   capillary pCO2
     --> (a.k.a.: systemic capillary pCO2)
     --> (a.k.a.: cap pCO2)
   CO2 excretion rate
     --> (a.k.a.: VCO2)
   dead space
     --> (a.k.a.: physiologic dead space)
     --> (a.k.a.: VD)
   FICO2
     --> (a.k.a.: fractionional inspired CO2)
   metabolic CO2 production rate
     --> (a.k.a.: CO2 prod)
   arterial pCO2
     --> (a.k.a.: pCO2)
     --> (a.k.a.: paCO2)
   respiratory rate
     --> (a.k.a.: RR)
   tidal volume
     --> (a.k.a.: VT)

Processes:
---------
   alveolocapillary diffusion
     --> (a.k.a.: a-c diff)
   bulk gas flow
     --> (a.k.a.: gas flow)
   the medullary chemoreflex
     --> (a.k.a.: chemoreflex)
     --> (a.k.a.: chemoref)
   circulatory CO2 transport
     --> (a.k.a.: CO2 trans)
   circulatory flow
     --> (a.k.a.: circ flow)
   CO2 elimination
     --> (a.k.a.: CO2 elim)
   lower airway gas mixing
     --> (a.k.a.: gas mixing)
   pulmonary gas exchange
```

```
    --> (a.k.a.: gas exch)
  pulmonary gas transport
    --> (a.k.a.: pulmonary ventilation)
    --> (a.k.a.: gas trans)
  respiratory control of pCO2
    --> (a.k.a.: respiratory pCO2 regulation)
    --> (a.k.a.: pCO2 regl)
  tissue-capillary CO2 diffusion
    --> (a.k.a.: t-c CO2 diff)

States:
------
  alveolar hyperventilation
  alveolar hypoventilation
  ARDS
    --> (a.k.a.: adult respiratory distress syndrome)
    --> (a.k.a.: shock lung)
  COPD
    --> (a.k.a.: chronic obstructive pulmonary disease)
  hypercapnea
    --> (a.k.a.: hypercarbia)
    --> (a.k.a.: hypoventilation)
  hypermetabolism
  hyperpnea
  hypocapnea
    --> (a.k.a.: hypocarbia)
    --> (a.k.a.: hyperventilation)
  hypometabolism
  hypopnea


Q:   What is arterial pCO2?

A:   Arterial pCO2 (pCO2, paCO2) is a physiologic parameter, the partial
pressure of carbon dioxide in arterial blood measured in mm Hg. Its normal
range is 38 to 42. Its physiologic range is 15 to 100. It is routine to
measure in most clinical settings. Increased arterial pCO2 is called
hypercapnea. Decreased arterial pCO2 is called hypocapnea. For more
information see West p. 1.
```

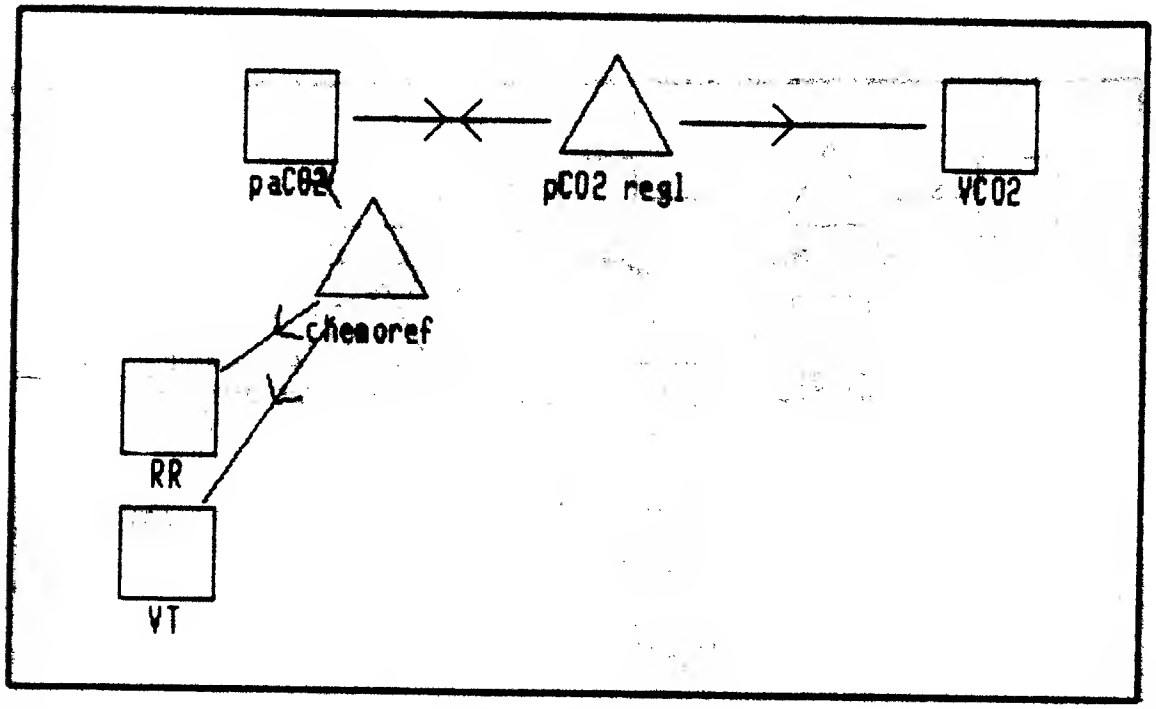
Q: What directly influences arterial pCO2 (pCO2, paCO2) ?

A: This question may be answered at three different levels of detail. At the most abstract level, arterial pCO2 is directly influenced by metebolic CO2 production rate through circulatory CO2 transport and arterial pCO2 through respiratory control of pCO2. At a more detailed level, arterial pCO2 is directly influenced by capillary pCO2 through circulatory flow and respiratory rate and tidal volume through pulmonary gas exchange. At an even more detailed level, arterial pCO2 is directly influenced by alveolar pCO2 and alveolocapillary CO2 diffusion rate through alveolocapillary diffusion.



Q: What is directly influenced by arterial pCO2 (pCO2, paCO2) ?

A: This question may be answered at two different levels of detail. At the most abstract level, arterial pCO2 directly influences CO2 excretion rate and arterial pCO2 through respiratory control of pCO2. At a more detailed level, arterial pCO2 directly influences respiratory rate and tidal volume through the medullary chemoreflex.

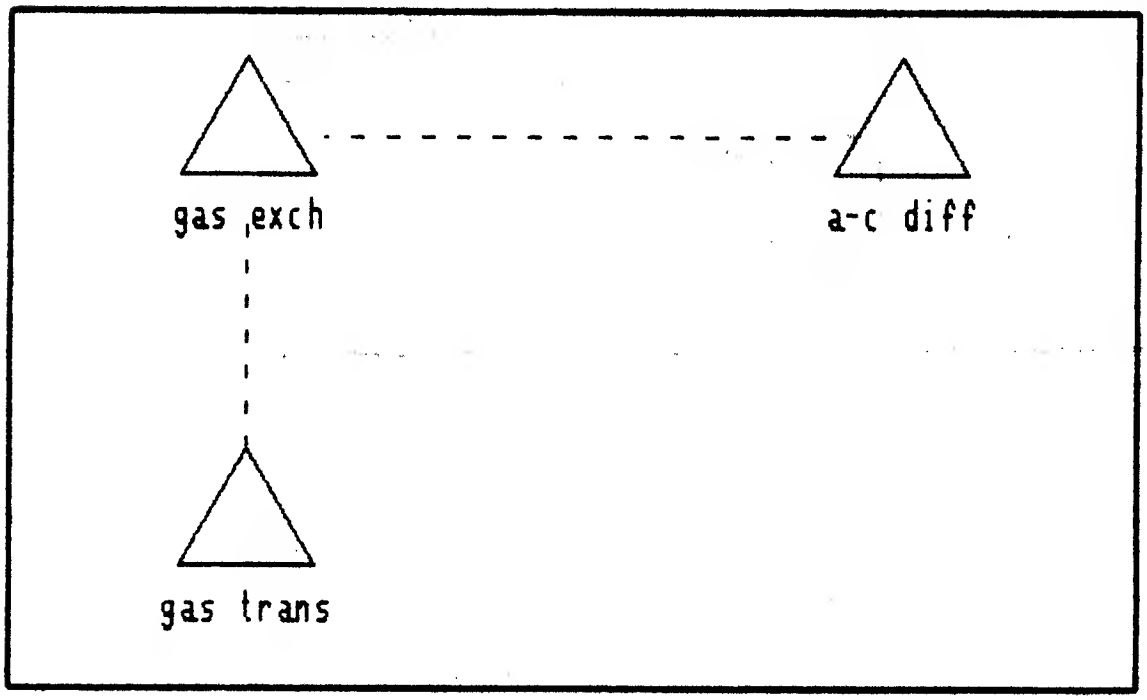


Q: What are the mechanisms of arterial pCO2?

A: Although this may be a reasonable question in a more general setting, it does not really make sense in the limited context of this model. Arterial pCO2 (pCO2, paCO2) is a physiologic parameter (an entity with a potentially masurable value) and does not have any mechanisms as such. In this modeling system only processes (defined as descriptions of the interaction between parameters) are said to have mechanisms. For example, one might ask about the mechanisms of tissue-capillary CO2 diffusion, which is a process. However, asking about the mechanisms of something like tidal volume (another parameter) or hyperpnea (a state) would not be meaningful.

Q: What are the mechanisms of pulmonary gas exchange?

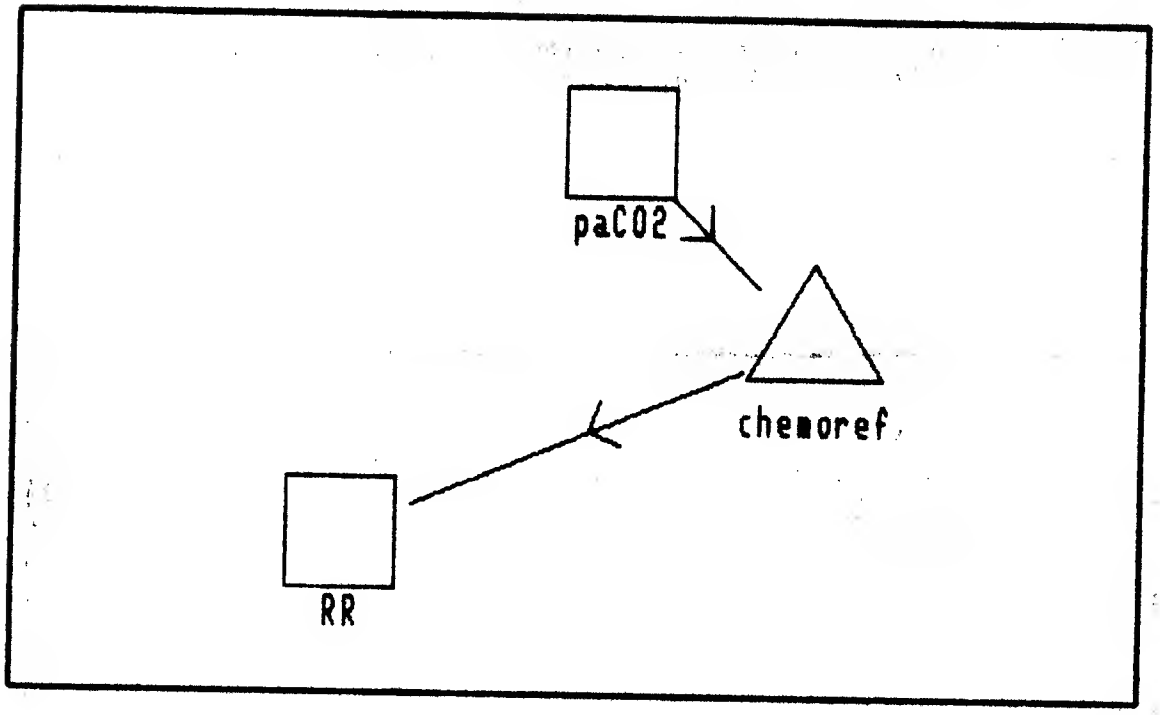
A: The mechanisms of pulmonary gas exchange (gas exch) are pulmonary gas transport and alveolocapillary diffusion.



APPENDIX III

Q: Does arterial pCO2 influence respiratory rate?

A: Yes, arterial pCO2 influences respiratory rate through the medullary chemoreflex.

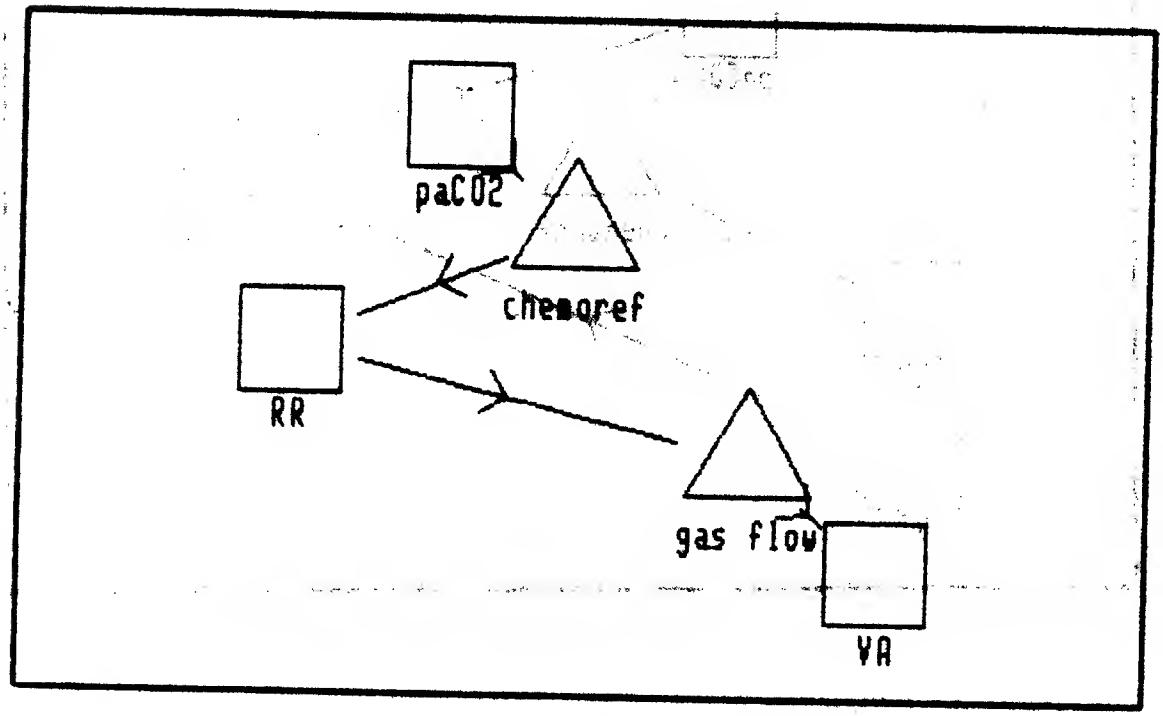


Q: Does arterial pCO2 influence metabolic CO2 production rate?

A: No, arterial pCO2 does not influence metabolic CO2 production rate.

Q: Does arterial pCO2 influence alveolar ventilation?

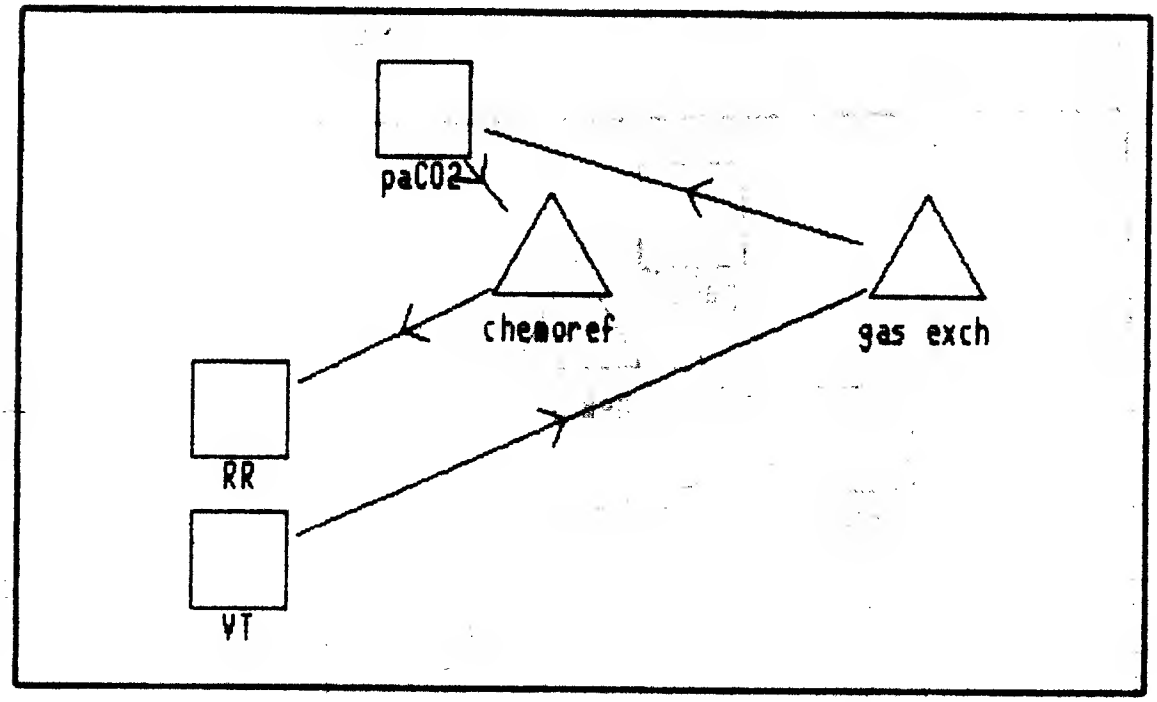
A: Not directly, however arterial pCO2 influences respiratory rate through the medullary chemoreflex and respiratory rate influences alveolar ventilation through bulk gas flow.



APPENDIX III

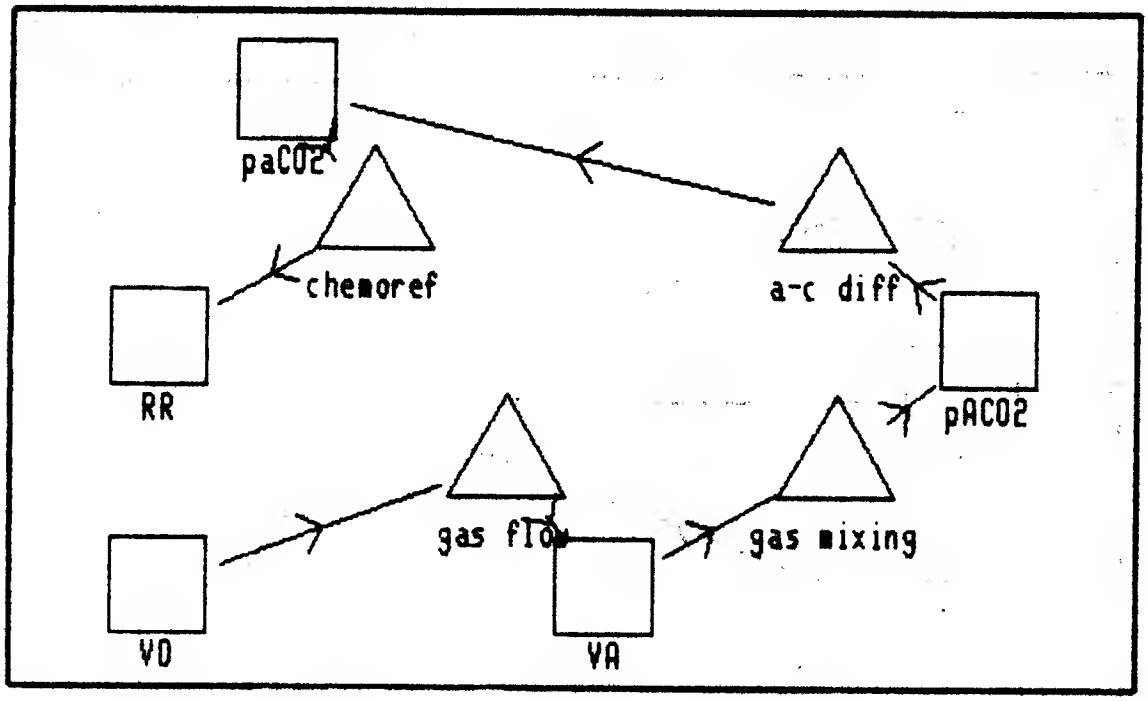
Q: Does tidal volume influence respiratory rate?

A: Not directly, however tidal volume influences arterial pCO2 through pulmonary gas exchange and arterial pCO2 influences respiratory rate through the medullary chemoreflex.



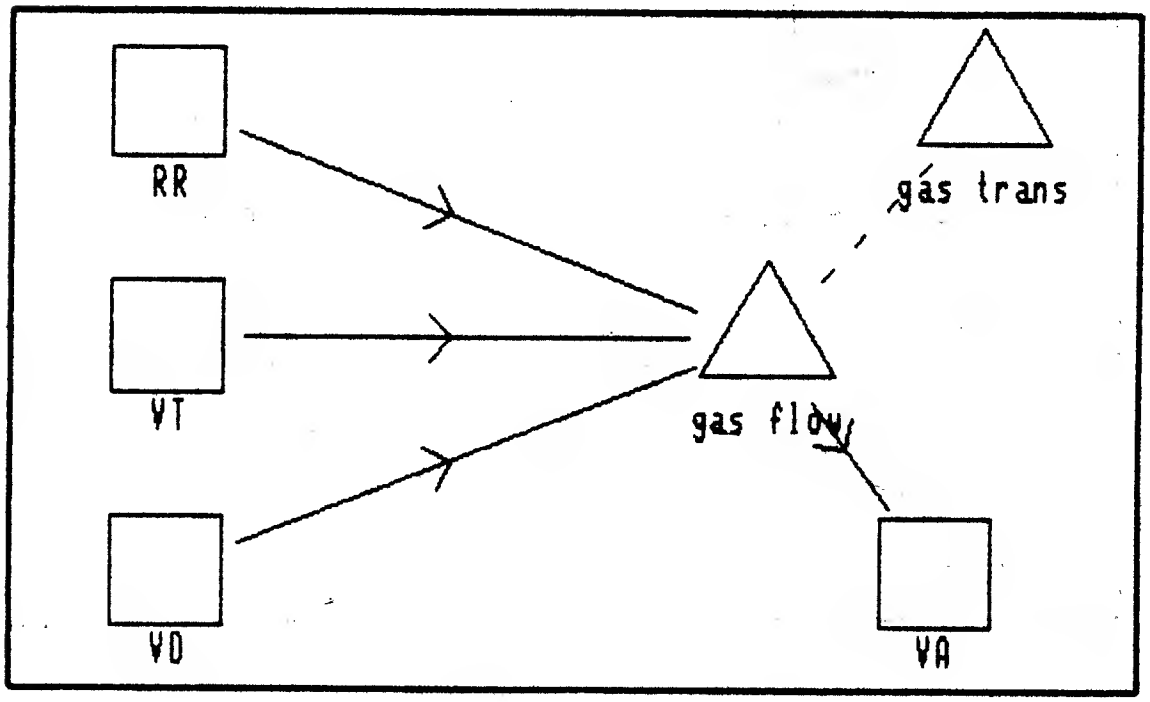
Q: Does dead space influence respiratory rate?

A: Not directly, however dead space influences alveolar ventilation through bulk gas flow, alveolar ventilation influences alveolar pCO2 through lower airway gas mixing, alveolar pCO2 influences arterial pCO2 through alveplocapillary diffusion and arterial pCO2 influences respiratory rate through the medullary chemoreflex.



Q: What is bulk gas flow?

A: Bulk gas flow (gas flow) is a physical process, the flow of gas volumes through the tracheobrochial tree. It is a mechanism of pulmonary gas transport. It mediates the influence of respiratory rate, tidal volume and dead space on alveolar ventilation. Increasing respiratory rate increases alveolar ventilation. Increasing tidal volume increases alveolar ventilation. Increasing dead space decreases alveolar ventilation. The following mathematical relationships apply: VA=RR*(VT-VD). Impaired bulk gas flow may be found in association with COPD. For more information see Guyton pp. 484-486 and West pp. 15-19.



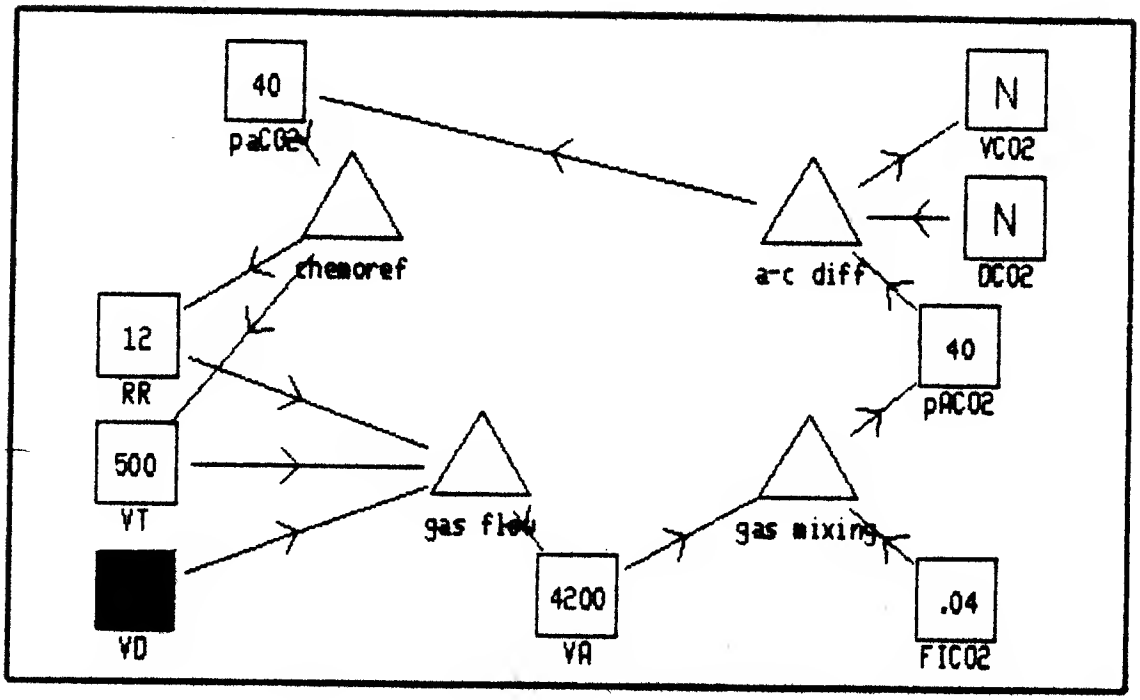
Q: What is ARDS?

A: ARDS (adult respiratory distress syndrome, shock lung) is a pathophysiologic state characterized by impaired alveolocapillary diffusion.

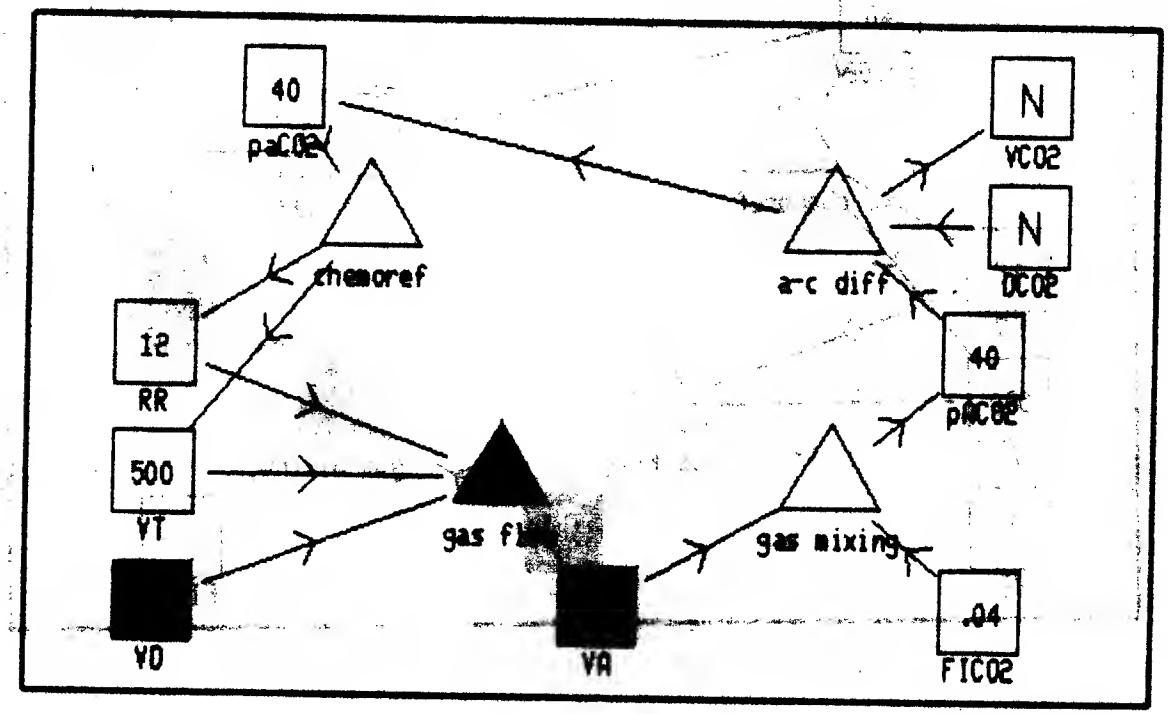
Q: What is hypocapnea?

A: Hypocapnea (hypocarbia, hyperventilation) is a physiologic state characterized by decreased arterial pCO2.

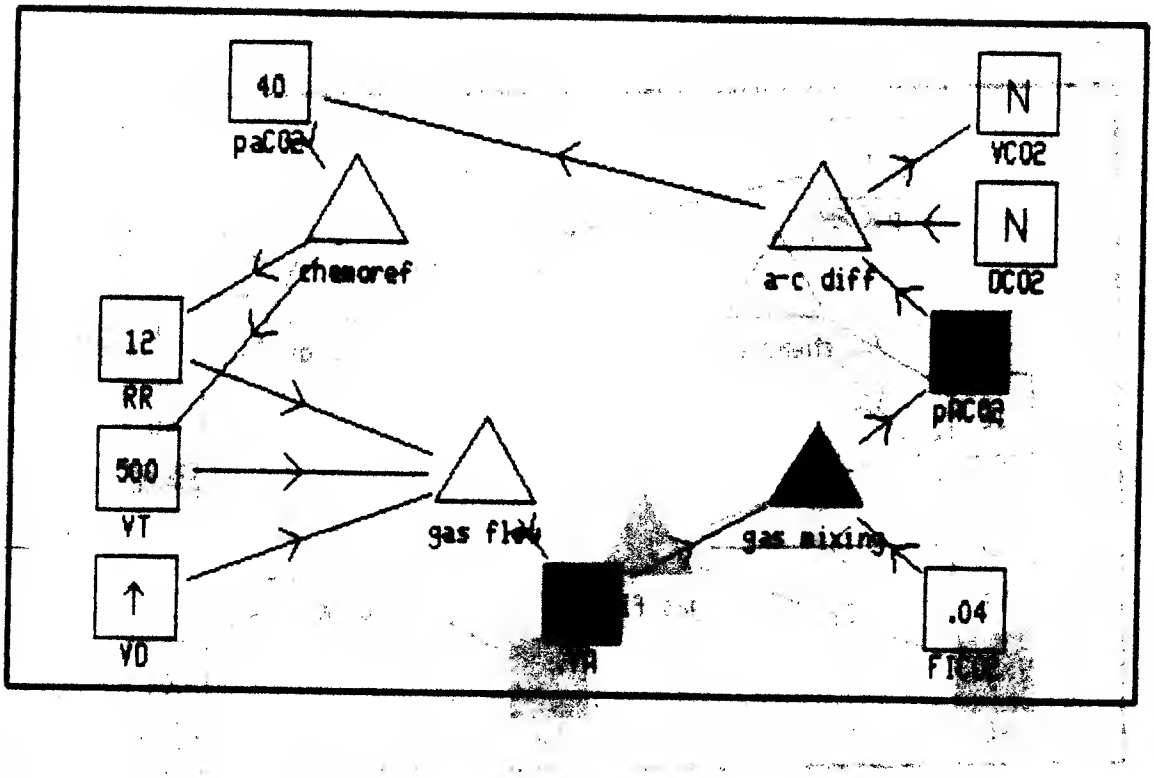
```
--- Start of Simulation ---
In step 1, dead space is increased as specified by the user.
```



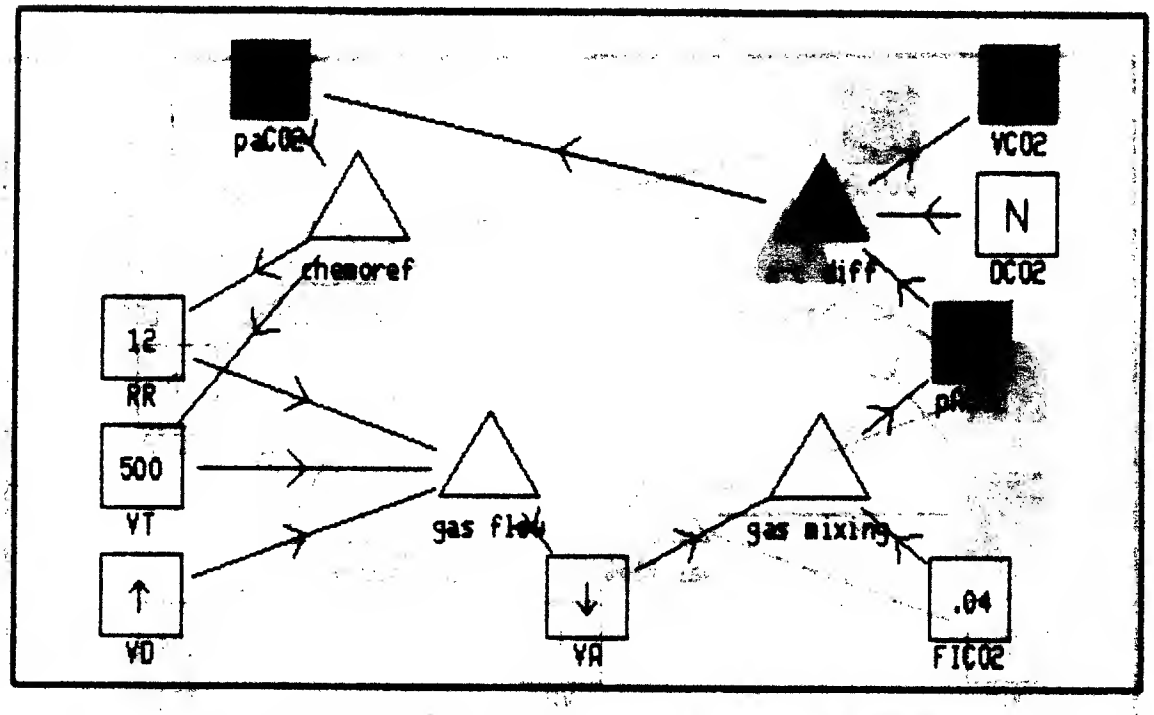
In step 2, alveolar ventilation is decreased [ie. alveolar hypoventilation] by increased dead space through bulk gas flow.



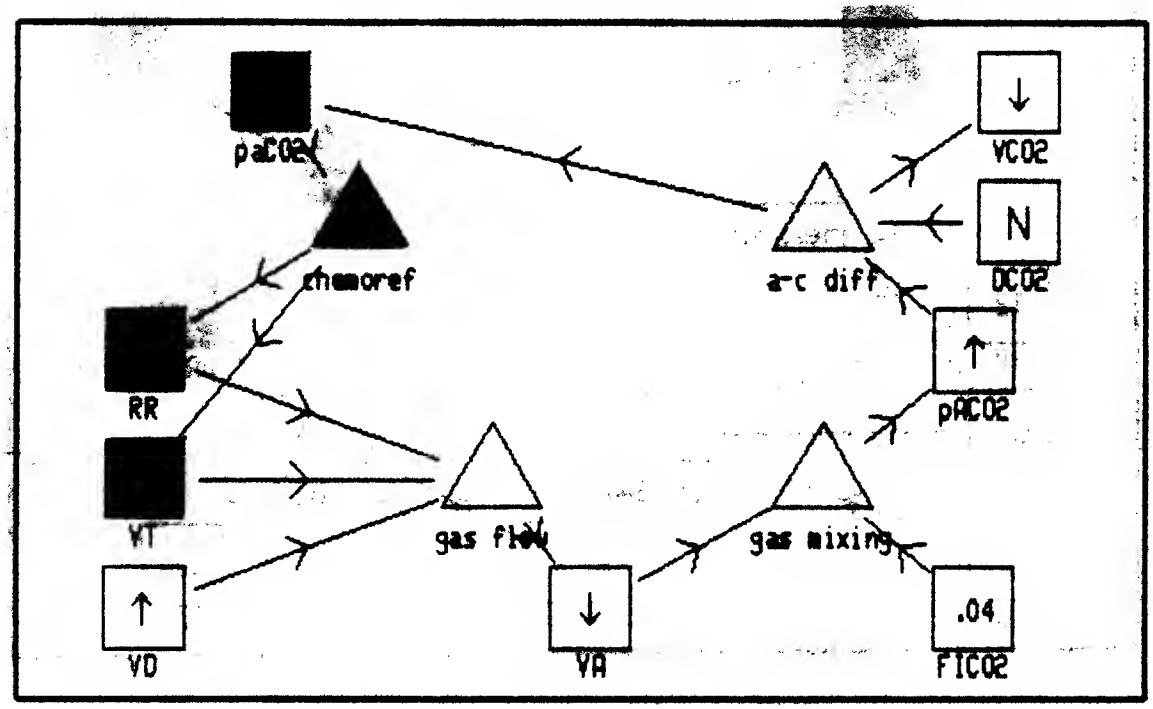
In step 3, alveolar pCO2 is increased by decreased alveolar ventilation through lower airway gas mixing.



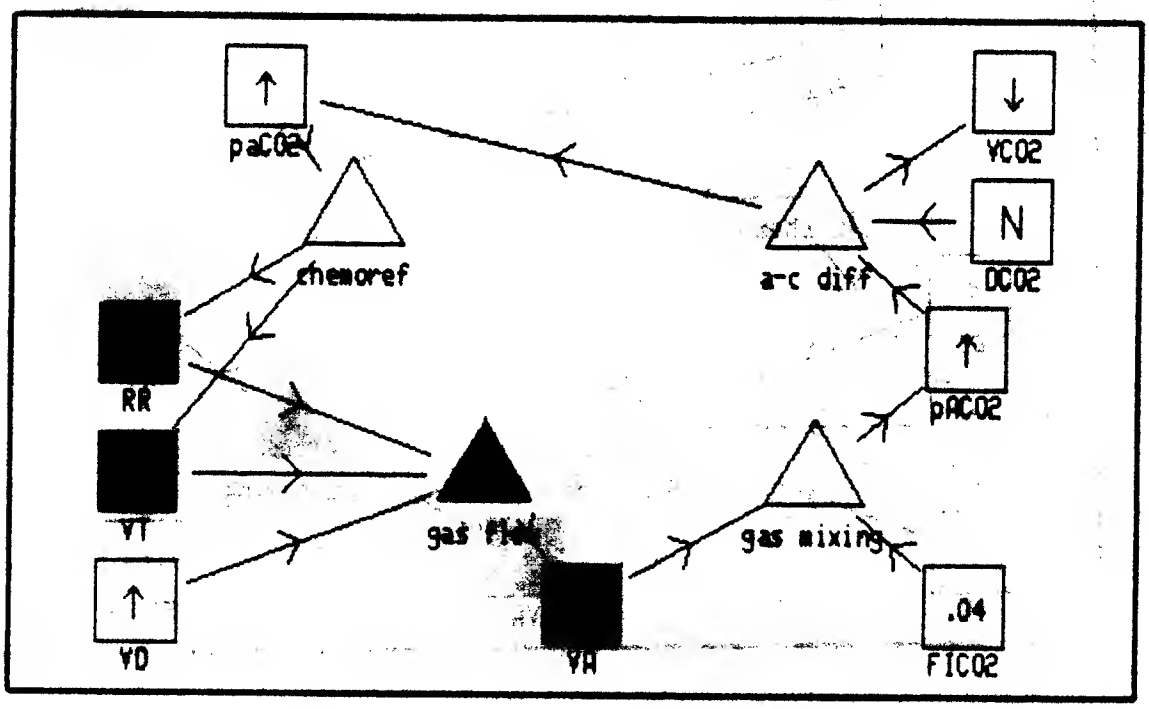
In step 4, CO2 excretion rate is decreased and arterial pCO2 is increased [ie. hypercapnea] by increased alveolar pCO2 through alveolocapillary diffusion.



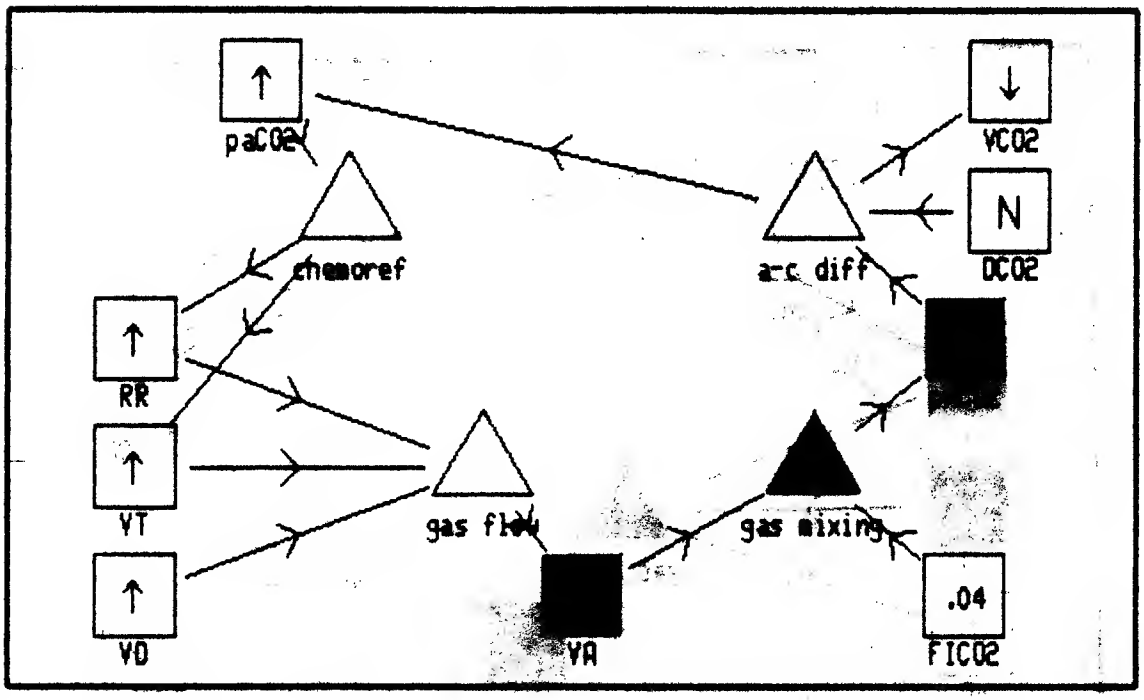
In step 5, respiratory rate is increased [ie. hyperpnea] and tidal volume is increased by increased arterial pCO2 through the medullary chemoreflex.



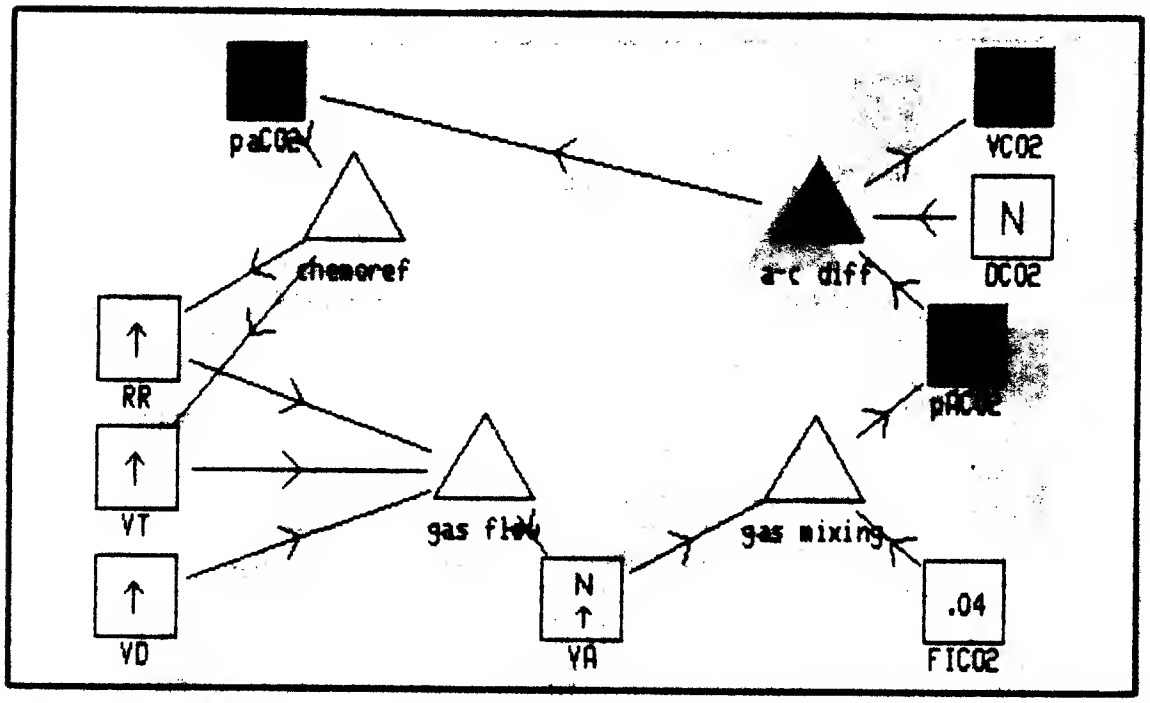
In step 6, alveolar ventilation is increased toward normal [ie. diminished alveolar hypoventilation] by increased respiratory rate and increased tidal volume through bulk gas flow.



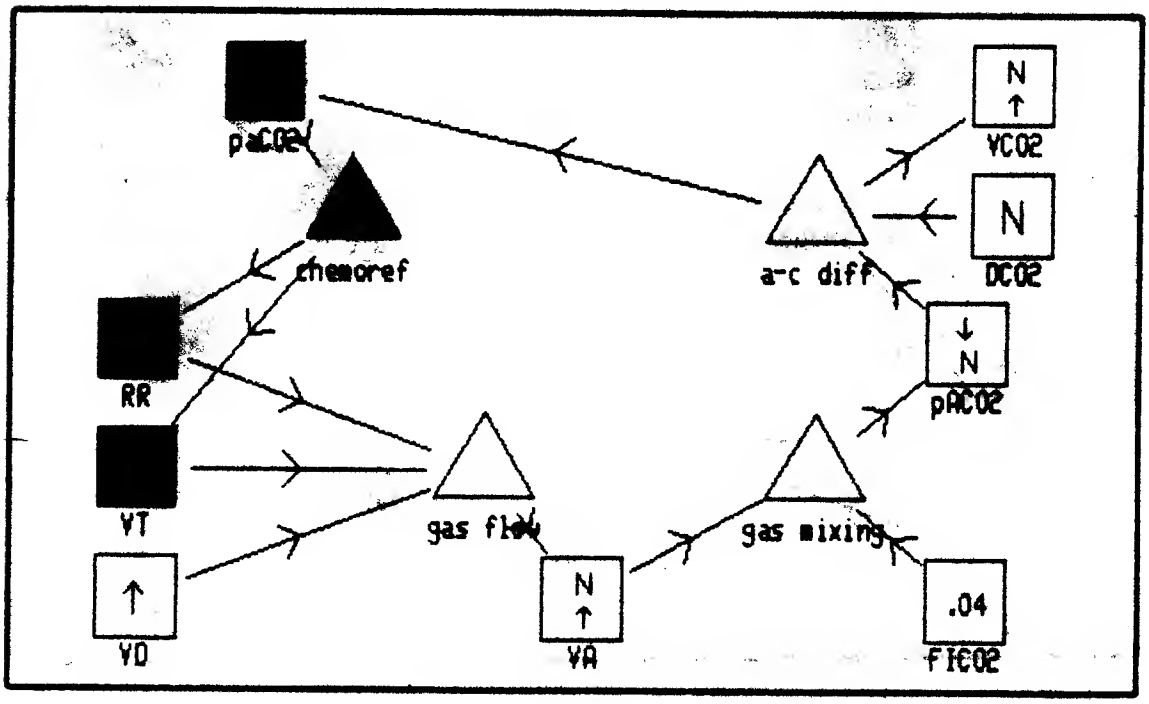
In step 7, alveolar pCO2 is decreased toward normal by increased alveolar ventilation through lower airway gas mixing.



In step 8, CO2 excretion rate is increased toward normal and arterial pCO2 is decreased toward normal [ie. diminished hypercapnea] by decreased alveolar pCO2 through alveolocapillary diffusion.

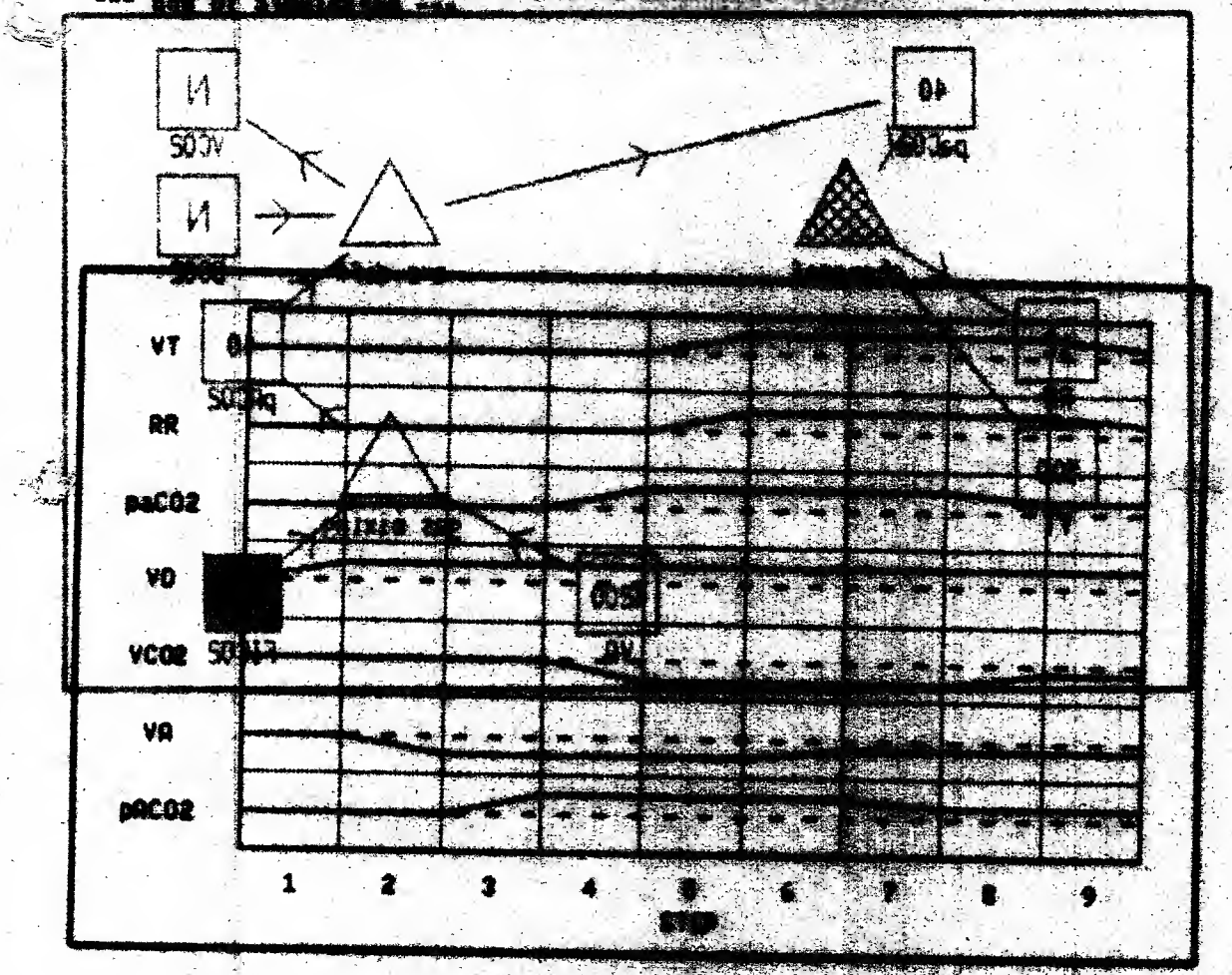


In step 9, respiratory rate is decreased toward normal [ie. diminished hyperpnea] and tidal volume is decreased toward normal by decreased arterial pCO2 through the medullary chemoreflex.
--- End of Simulation ---



--- Summary: Start of Simulation ---

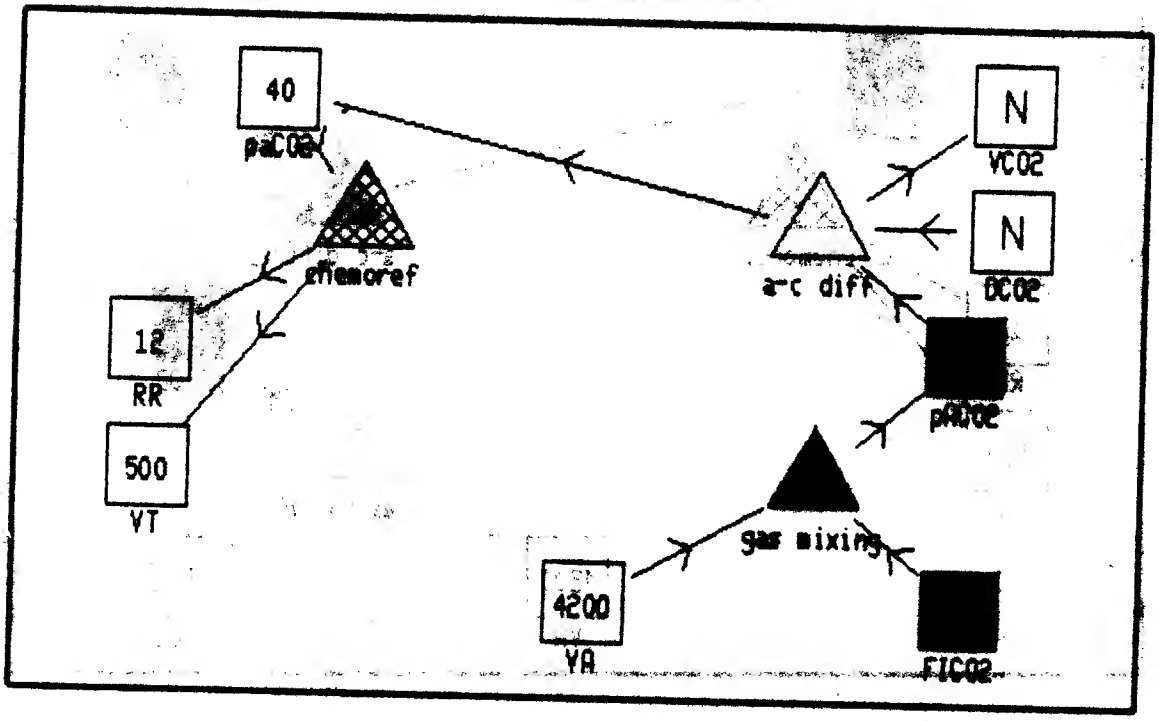
Step 1: FiCO2 is increased and the resulting alveolar ventilation is increased [to. alveolar hyperventilation].
Step 2: Alveolar ventilation is increased [to. alveolar hyperventilation].
Step 3: Alveolar pCO2 is decreased.
Step 4: CO2 excretion rate is decreased and arterial pCO2 is increased [to. hypercapnea].
Step 5: Respiratory rate is increased [to. hyperpnea] and tidal volume is increased.
Step 6: Alveolar ventilation is increased toward normal [to. diminished alveolar hyperventilation].
Step 7: Alveolar pCO2 is decreased toward normal.
Step 8: CO2 excretion rate is increased toward normal and arterial pCO2 is decreased toward normal [to. diminished
Step 9: Respiratory rate is decreased [to. diminished hyperpnea] and tidal volume is decreased toward normal.
--- End of Simulation ---



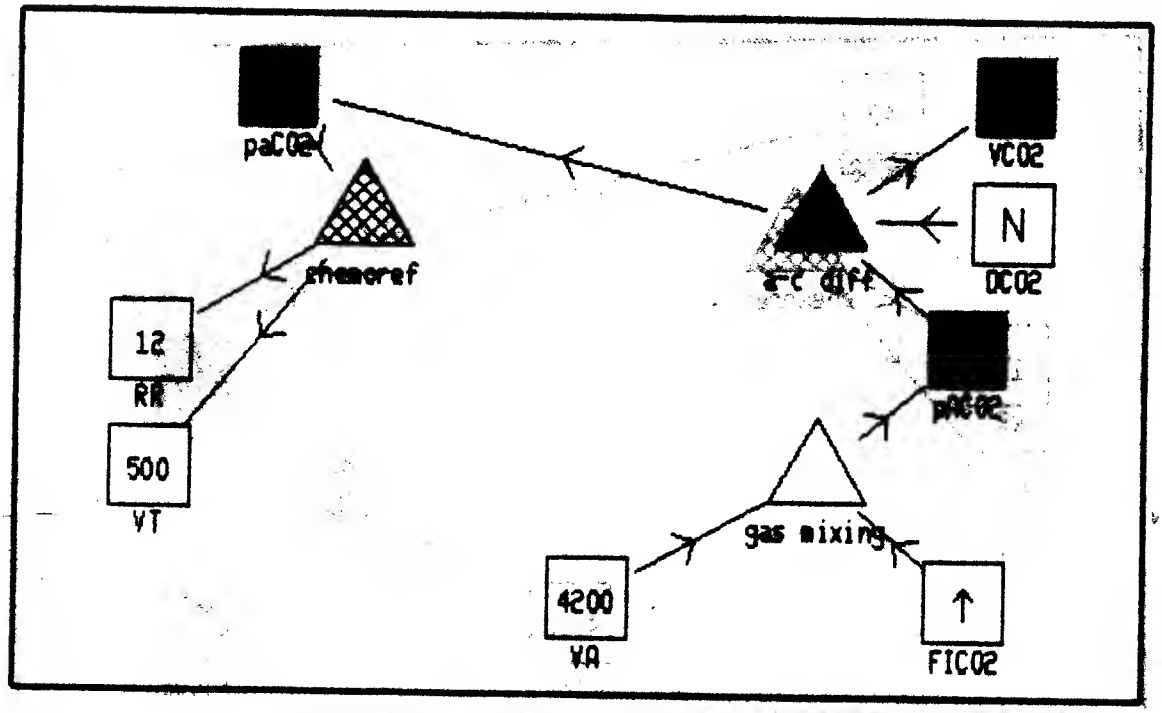
--- Start of Simulation ---

In step 1, FECO2 is increased and the [illegible]
specified by the user.

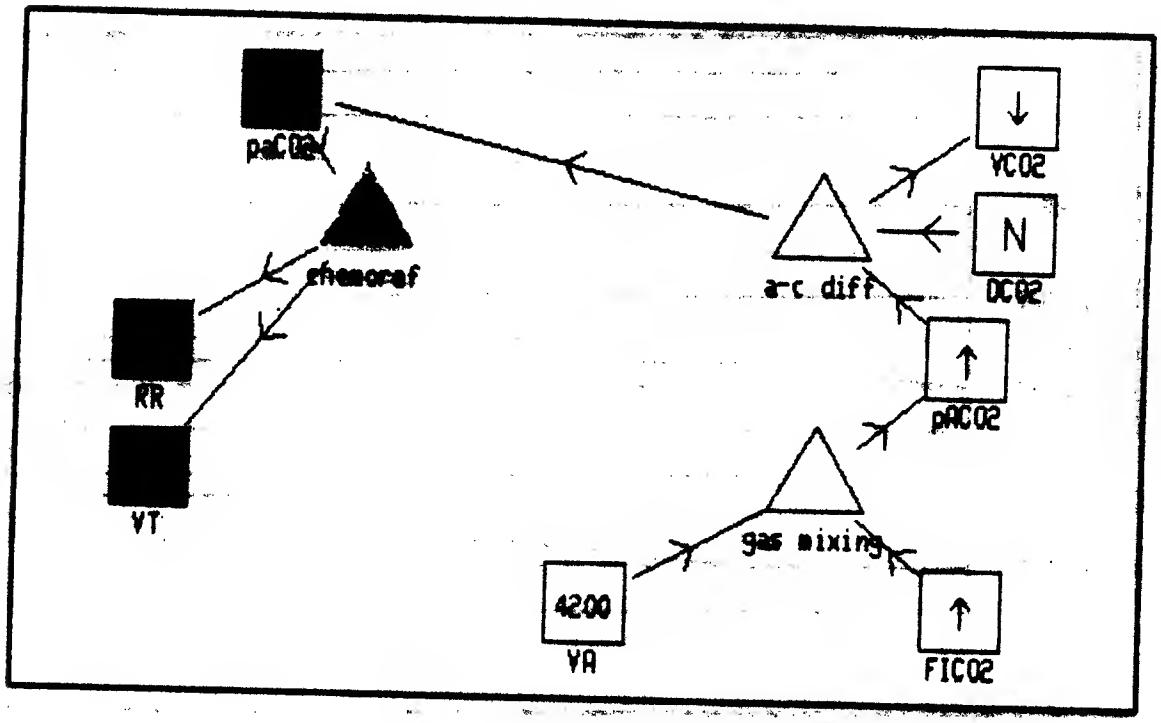
In step 2, alveolar pCO2 is increased by increased FICO2 through lower airway gas mixing.



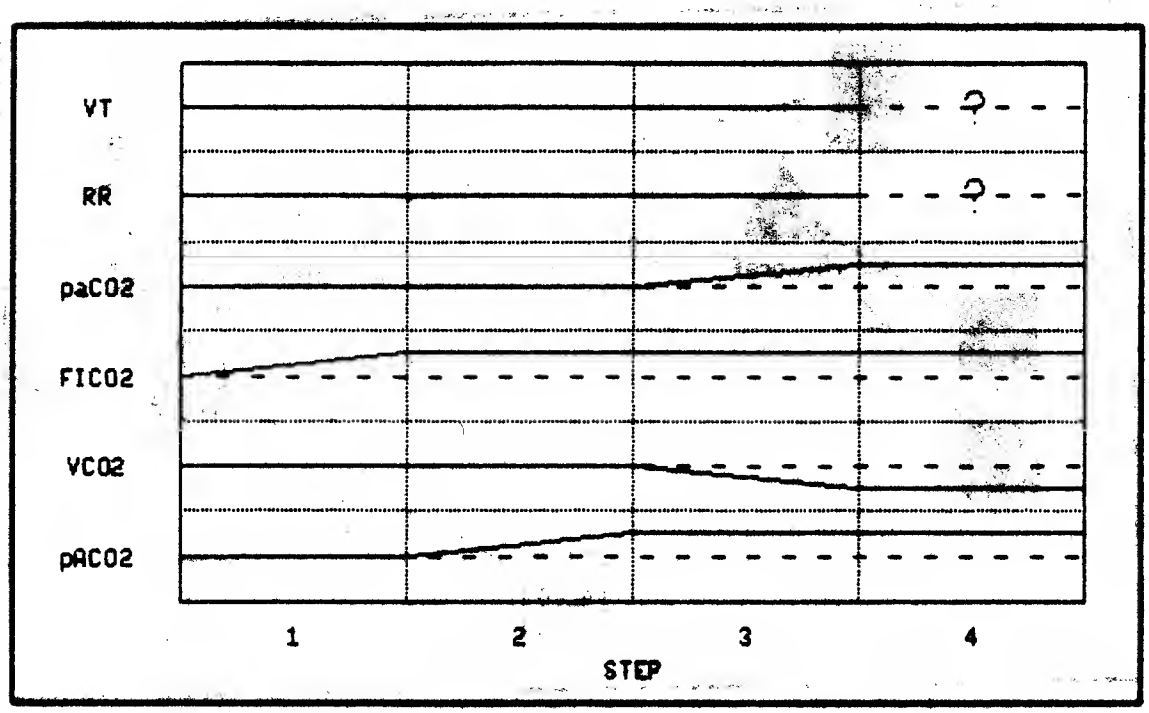
In step 3, CO2 excretion rate is decreased and arterial pCO2 is increased [ie. hypercapnea] by increased alveolar pCO2 through alveolocapillary diffusion.



Ordinarily, respiratory rate would be increased [ie. hyperpnea] and tidal volume would be increased by increased arterial pCO2 through the medullary chemoreflex. But in step 4, the medullary chemoreflex is impaired and cannot mediate the influence of increased arterial pCO2. Therefore respiratory rate is unknown and tidal volume is unknown.
--- End of Simulation ---



```
--- Summary: Start of Simulation ---
Step 1:  FICO2 is increased and the medullary chemoreflex is impaired.
Step 2:  Alveolar pCO2 is increased.
Step 3:  CO2 excretion rate is decreased and arterial pCO2 is increased [ie.
hypercapnea].
Step 4:  Respiratory rate is unknown and tidal volume is unknown.
--- End of Simulation ---
```



Appendix IV
Evaluation Instruments

The following are the educational objectives, homework assignment, and quiz which were distributed to the New Pathway students, faculty, and staff as part of the preliminary evaluation of KBPMS.

Educational Objectives

The goal of the Respiratory Physiology V1.1 model is to provide a medium for exploration of selected aspects of carbon dioxide homeostasis by the respiratory system. After using this model to carry out an appropriate homework assigment, within the framework of a comprehensive human physiology course, students should be able to:

1) Understand, define, and appropriately use the following terms:

   alveolar pCO_2 ($pACO_2$)
   alveolar ventilation (VA)
   alveolocapillary CO_2 diffusion rate (DCO_2)
   capillary pCO_2
   CO_2 excretion rate (VCO_2)
   dead space (VD)
   fractional inspired CO_2 ($FICO_2$)
   metabolic CO_2 production rate
   arterial pCO_2 ($paCO_2$)
   respiratory rate (RR)
   tidal volume (VT)
   alveolocapillary diffusion
   bulk gas flow
   circulatory CO_2 transport
   circulatory flow
   the medullary chemoreflex
   lower airway gas mixing
   pulmonary gas exchange
   pulmonary gas transport
   pulmonary ventilation
   respiratory control of pCO_2
   tissue-capillary CO_2 diffusion
   alveolar hyperventilation
   alveolar hypoventilation
   hypercapnea
   hypercarbia
   hyperventilation
   hyperpnea
   hypocapnea
   hypocarbia
   hypoventilation
   hypopnea

2) Understand the input/output relationship of metabolic CO_2 production and respiratory CO_2 excretion.

3) Understand the feedback loop which governs respiratory control of CO_2, the role of the variaous physiologic parameters and processes which comprise this loop, and the central importance of $paCO_2$ and medullary chemoreflexes.

4) Understand some potential sources of dysfunction in respiratory CO_2 homeostasis and the clinical situations with which they may be associated.

The Homework Assignment

From: Dr. Martin Kushmerick
To: New Pathway Students
Date: January 10, 1988

Robert Kunstaetter, M.D., working with Octo Barnett and the Lab of Computer Science at M.I.T. has prepared a learning module about carbon dioxide homeostasis for the HP-150. My belief is that this is a very good example of the use of a computer as a learning tool. I ask you therefore to participate enthusiastically in the following plan. Although we are quite convinced that the program is useful, we want to confirm this intuition, and find out whether in fact this program is useful to you. First you will work on a homework exercise, described below. Later (on Tuesday or Wednesday, Jan. 14 or 15) there will be a take-home "quiz".

In order to find out to what extent the computer program is convenient and useful, and to what extent it actually aids learning, I want you all to perform a special study assignment to explore certain aspects of carbon dioxide homeostasis. The problems to work on are obviously a subset of the work you are doing in respect to the case of Mr. Allen, and closely relate to the respiratory laboratory. Thus consider each of the following questions. Realizing the pressure of work, I am not asking you to write up anything for this assignment, but please give it careful consideration none-the-less. In addition, please keep a record of the amount of time you devoted to various resources (textbooks, lab material, computer program, journals, etc.) in doing this exercise and hand this in to me.

Please consider the following six questions:

1) How does the respiratory system respond to changing amounts of carbon dioxide in the body?

2) What physiologic pathways mediate this response?

3) If CO_2 homeostasis is viewed as a feedback loop, what are the sensors, effectors, mediators, and setpoints of this feedback system?

4) How might the pathways of CO_2 homeostasis be disrupted so as to impair this physiologic response?

5) What will happen to respiration if you rebreathe from closed bag? Consider a bag that initially contains 10 L of room air. Consider the changes in its volume and the partial pressures of CO_2 and O2.

6) Why does ventilation increase during exercise, and what effect will this have on alveolar gas tensions?

Now, regarding the evaluation of the computer program as a learning tool. Only half of you (Groups A & B) will be given the

computer program at this time. I ask this group to use it extensively as an important learning resource along with your texts and laboratory materials. I ask both groups to thoughtfully go through the above homework exercise. On Tuesday or Wednesday everyone will be given a take-home "quiz" which is intended to evaluate the your overall understanding of CO_2 homeosatsis. This "quiz" should be done closed-book and should take no more than 1.5 hours of your time. It will in no way influence your overall evaluation in this course. Following the "quiz", the other half of the class will have access to the computer program.

If you have any questions concerning the use of the computer program, please contact Robert Kunstaetter via HP-Desk or call him at home. Your evaluation of the program is crucial to our continuing effort to provide learning resources of this type. Everyone should submit an assesment of the program's strengths, weaknesses, and suggestions for specific changes to Dr. Kunstaetter via HP-Desk.

The Quiz

Please work alone and without notes, books or use of the computer. Complete your work within 1 and a half hours, i.e. about 10 minutes per question. Please answer each of the following questions in one or two paragraphs:

1) What is dead space?

2) How do tidal volume, respiratory rate, dead space, and alveolar ventilation interact?

3) What directly influences alveolar pCO_2?

4) Arterial pCO_2 plays a central role in carbon dioxide homeostasis by the respiratory system. Explain.

5) Does dead space influence respiratory rate? Explain.

6) What would be the consequence of impaired medullary chemoreflexes in an otherwise healthy individual?

7) Describe the physiologic events which would take place if a healthy person was to breathe air which had a higher than normal concentration of CO_2.

8) A patient in the Intensive Care Unit is intubated, paralysed, and being artificially ventilated with 30% O2 at a rate of 18 breaths/minute, with a tidal volume of 750 mls and a dead space of 200 mls. His last arterial blood gas showed: pO2=70, pCO_2=30, pH=7.51, [HCO3]=23. You are concerned about his respiratory alkalosis and borderline oxygenation. You can control the patient's tidal volume and respiratory rate by adjusting the settings on his ventilator and you can control his dead space by adjusting the length of tubing connecting him to the ventiltor. You consider increasing his tidal volume to one liter but the respiratory therapist on duty reminds you that the patient has severe emphysema and that the increaaed pressure might rupture a bleb in his lung and cause a pneumothorax. What else might you do to decrease the alkalosis and increase oxygenation?

APPENDIX V

Appendix V
Evaluation Raw Data

The following are the scores of the two groups of New Pathways students on the evaluation quiz:

(all scores are %)

		Experimental					Control		
Student --->		1	2	3	4	5	6	7	8
Question	Type								
1	I	50	90	90	100	50	100	100	80
2	II	20	100	20	90	100	20	20	90
3	I	75	50	75	100	75	100	50	50
4	II	100	90	80	100	80	90	100	100
5	I	100	100	50	100	50	100	80	50
6	II	20	100	100	50	100	100	100	100
7	I	90	90	80	100	90	90	90	100
8	II	100	40	60	80	80	20	80	20
All Type I		79	83	74	100	66	98	80	70
All Type II		60	83	65	80	90	58	75	78
All Questions		69	83	69	90	78	78	78	74

APPENDIX VI

Appendix VI
Student Comments

The following are some of the New Pathway students' comments regarding KBPMS. They are unedited except that the students' names have been omitted.

KUNSTAETTER,ROBERT / NP/1 - HPDESK print.

Message. Dated: 01/16/86 at 1719.
Subject: Computer teaching

The CO2 program loaded without a hitch. How about the program itself. I spent about two hours running the program, and I wasn't really satisfied with the amount I learned. The graphics were great, the explanations were amazing, but the simulations really left me cold.

I first spent time in the explanations section, getting oriented to the terms, and relations on the big diagram. Seeing the heirarchy of processes was interesting. I usually love to see a complex body of interrelationships mapped out like this, but CO2 homeostasis just isn't complicated enough for this to be worth it. I think I already understood a majority of the relationships. (Now if you worked some other stuff into this model, like O2, pH, water and electrolyte homeostasis, THEN we'd be talking a useful model.)

The simulations were frustrating. First, you can't simulate pathology because impairing a process interrupts the simulation if it's in the path of changes, and if the process isn't in the path of changes, impairing it does nothing. Second, the thing isn't quantative. Irt

This proved to be especially disappointing after entering specific altered values. When I tried altering CO2 production values, it was happy to call both .0000001 and 100000 normal.

In the final analysis, the program was really beautiful, but I think I someone could have explained everything itteught me in about ten minutes.

KUNSTAETTER,ROBERT / NP/1 - HPDESK print.

Message. Dated: 01/23/86 at 1004.
Subject: resp prog

Yea, though I hang my head in shame, still will I testify to the strengths and weaknesses of the program. I finally got a chance to really work through it late last night. If pressed, I will do the exercise, but in the meantime I'd like to tell tou what I thought about USING it. First, I didn't find it what you might call 'user friendly'. I mean, what is a parameter anyway? It took me three shots at the program to understand how to get around inside of it well enough to really exploit it. Second, I don't think it is a useful tool for learning, which isn't to say that it won't be great for review. I preferred the format of the acid/base program in this regard. I guess I feel more comfortable when led through an exercise on material which is not very clear to me. Once I finally figured out how to use the program, I realized that there really is a lot of iion inside of it. Again, however, I don't think that I could really have used all of that info if I was just beginning to study respiration. Also, the terseness of the language in the explanations makes this a really heavy exercise, demanding a lot of energy just in figuring out how things are said. Again, I contrast this with the acid/base program which, though itself limited, presents a generally more accessible exercise. Finally, I would recommend (if it is possible) that a way be found to make changes in a simulation while it is being studied. I found myself wishing that I could see the normal relationship between things in front of me before I started perturbing the works. I also wished I could play some compensation games while inside a simulation. I realize that some of my problems might have something to do with my not feeling comfortable with or understanding how to move around inside of the program, but that may be a design problem to consider as well. Anyway, I hope these musings are of some value to you. I apologize for not being the most accomodating in helping out with the testing of the program; I hope that my comments here accomplish something along the lines of what you are interested in hearing about.

KUNSTAETTER,ROBERT / NP/1 - HPDESK print.

Message. Dated: 01/23/86 at 2356.
Subject: Respiratory

I finally get to you on the Respiratory program. My initial impression was that it wasn't particularly valuable. I felt that the model was not teaching me anything that I didn't already know and that the format was somewhat forced. Then I finally found the time to take the exam, and here are my new thoughts.

First of all, I was irritated about having to spend the time on the exam, but it turned out to be a wonderfully integrating experience for me. It forced me to organize what I had learned and identified what I had not. Interestingly, as I thought about answering the questions, the simulation diagram k popping in my head and it helped me to very clearly plan the flow of my ideas. I now realize that the repetition was more valuable than I had suspected! I was unclear about the role of peripheral chemoreceptors, however, and am not sure that they were included in the program.

Learning by computer certainly is fun and a pleasant break from the more passive reading. Good luck in future development. It is unlikely, however, that any given program will be equally helpful to all.